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CONCERNING THE "VITALITY" OF THE CALCIFIED DENTAL TISSUES[1]

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INTRODUCTION

The vitality of the enamel, dentin, and cementum is still an open question. Up to the end of the last century, dental tissues were believed to possess a low degree of vitality and hence were considered subject to systemic metabolic changes. Later, there was a complete reversal of opinion, resulting in the almost general belief that the enamel was an inert, dead structure; some authorities going so far as to include the dentin and the cementum. This point of view seems to carry the non-vitalist conception to the extreme. It is true that both dentin and cementum are composed principally of a matrix, nevertheless cells or cell processes are included in these tissues. The belief that enamel is an inert structure has its justification in the fact that its formative organ is destroyed upon the completion of the crown of the tooth; therefore, no *new* enamel can be formed. But this fact does not necessarily exclude the possibilities of physical and chemical changes occurring in the enamel after eruption of the tooth.

The conception of non-vitality of the dental tissues is slowly undergoing a second reversal, brought about by the observation of the beneficial effect of diet on the teeth. It seems that the entire question of the vitality of the teeth rests upon an exact definition of terms. Churchill (1) presents his conception of the subject in a clear and concise manner. He states:

> In view of our lack of knowledge concerning vital processes in general, it seems difficult to offer an opinion in a controversy with regard to the vitality of the hard structures of the teeth. And yet, this question is so often the subject of discussion in professional circles that a commitment does not appear out of place.

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[1] Presented at the 15th General Meeting of the International Association for Dental Research, Baltimore, March 14, 1937.

As we attempt to penetrate into the secrets of our tissues and try to explain their hidden functions, we gradually drift away from dogmatic assertion.

We recognize vitality in the body cells by their performance of functions we conventionally regard as characteristic of life. However, we find it difficult to accept the view that these cells possess a monopoly in regard to such vital phenomena.

We might ask: Is metabolism limited to the cellular elements of the body? It is important to point out that, to some extent, everywhere in our tissues chemical changes occur which in a broader sense may be called metabolic. The body fluids, such as the lymph, carry the products of cell activities to and from the intercellular substance, adding and subtracting in accord with osmotic and utilitarian conditions. This fact together with the interdependence of cells and ground substance, broadens our concept of vitality. In this wider concept, anabolism, which is a synthetic process, and catabolism, which is analytic in nature, are factors which, in addition to intracellular processes, are components of metabolism.

If the hard structures are regarded as vital structures, then the following question must be answered: What is contributed by the pulp and the dental periostium to the vitality, in particular the functional characteristics of the tooth?

. . . . . .

Although certain facts point to the probability that in a broad sense enamel partakes of metabolic changes, such changes have in our estimation not as yet been incontrovertibly proven. Whatever processes in the broadest physiologic sense do occur, must be extremely slow in this densest of human structures. Experimental evidence points to changes brought about from within rather than from without.

. . . . . .

Summarizing, we may say that the question of vitality of the hard structures of the teeth is decided by definitions.

Those who believe that vitality is dependent on the presence of phenomena characteristic of cell life as metabolism, respiration, growth, regeneration, reproduction, irritability, contractility, and conductivity, naturally would regard the hard tooth structures as non-vital.

However, those who would consider the physical and chemical changes in the intercellular substance as representative of vital activities in the broader sense, will regard enamel, dentin, and cementum as participating in the vital processes of the body. We tend towards the later point of view.

Our observations indicate that the calcified dental tissues are vital in the broad sense that physical and chemical changes do occur in their intercellular substances after the completion and eruption of the teeth. The experiments described in this paper present evidence of channels in the dentin and enamel which contain a fluid emanating from the dental pulp. It is probable that the physical changes observed in these tissues are caused by the agency of this fluid.

HISTORICAL

The idea that the enamel and dentin are non-vital, is based on the belief that they are non-vascular and that the enamel is an imper-

meable, wholly mineral structure. Contrary evidence has been presented by Bodecker (2) Chase (3), Malleson (4), Rosebury (5), and others who showed that enamel contains organic matter; Bodecker and Gies (6) defined this as a protein. Beust (7) was the first to force stains through enamel in vitro, while Prinz (8), Fish (9) and Bertram (10) demonstrated dye diffusion in vital teeth. Bunting and Rickert (11), Klein and Amberson (12), and Applebaum (13) demonstrated the permeability of the enamel by electro-endosmotic means. Applebaum (14) defined the presence of a stain in enamel, originating from an amalgam filling, proving that this structure was permeable during life. Hollander (15) et al showed a variation in the calcification of the enamel, corresponding to the bands of Schreger. LeFevre and Hodge (16) suggested that calcification of the dentin continues after the eruption of the fully formed tooth. Karlstrom (17) offer similar evidence for the enamel and showed by means of hardness tests that calcification is reduced in cases of mal-nutritional pregnancy. Beust (18), on the basis of his staining technique, had long considered that an increase in mineral matter occurs in the teeth after their eruption; he named this process—maturation. Applebaum (19) showed by means of the Grenz ray that an increase in calcification in the dentin occurs in those portions which are subjected to the irritation of dental caries. Further in his study of the incipient lesion of caries of the enamel, he demonstrated that variations in density in this tissue are related to the varying speed of caries penetration. Clinical and experimental evidence therefore, indicate that the dentin and enamel are affected by physiological activity after the eruption of the teeth. Nevertheless, the physical and chemical changes, noted in the intercellular substances of the dentin and enamel, are not generally attributed to vital activity.

Much depends on the answer to this question. It is true that most biologically trained observers do not doubt the vitality of any normal tissue retained permanently in the body. Nevertheless, it seems necessary to advance further proof, to entirely convince the dental profession and also to make further progress toward a solution of the causes of dental caries and periodontoclasia.

The importance of defining the status of the dental tissues in relation to possible physiological activity is apparent. Some of the data

necessary to further define this problem are: 1) Is a fluid present in the dentin and enamel? 2) If so, what is its origin? 3) What rôle does the vitality of the dental pulp play? 4) Is there any difference in the behavior of a fluid in the calcified dental tissues (enamel, dentin, and cementum) in vital and devitalized teeth?

Prinz (8), Fish (9), and Bertram (10) have shown the way in which these data can be obtained. Prinz seems to have been the first to drill an experimental cavity into a vital tooth and insert a dye. His purpose was primarily to show the destructive action of formaldehyde on the dentin and the pulp. He added one per cent methylene blue to the formaldehyde; both reagents penetrated deeply, so that the pulp died in three days and traces of dye could be found in the canal. The following experiments on animal and human teeth were made which corroborate and extend the earlier investigations of Prinz, Fish and Bertram. A preliminary report (20) of this work was published describing the variable penetration of the Hartman desensitizing solution.

METHOD

Forty-two teeth of dogs, thirteen of monkeys, and twenty-four of man were used in these experiments.[2] The operations described below are shown in a diagram (*fig. 13c*). The animals were anesthetized with ether. Each tooth selected for experimentation was dried with warm air and isolated with sponges and cotton rolls to avoid the penetration of the saliva into the operating field. A small cavity was cut into the labial surface of the tooth with a No. 1 round bur, for about half the depth of the crown, care being taken not to expose the pulp. The cavity was then cleaned with a blast of air to remove any particles of dentin or enamel. In human and dog teeth, a dry dye was packed into the lower portion of the cavity so that it came in contact only with the dentin; in monkeys the dye was used in the form of an aqueous paste. The dyes employed in these experiments were: water-soluble eosin, congo-red, borax carmine, argyrol, silver nitrate crystals, and potassium permanganate. Oxy-phosphate cement was used to seal the cavity. The monkeys were kept alive for fourteen days, whereas the termination of the experiments on dogs varied from one to twenty-eight days. In humans, the specimens were taken seven to forty days after insertion of the stain.

Sections of the jaws containing the stained teeth were immediately removed from the sacrificed animal without fixing. One half of each tooth was ground off as nearly parallel as possible to the pulp canal and the dye deposit. This was done under oil and no other fluid came into contact with the specimens up to the time of taking the first photograph. This technique excluded the possibility of the stain being spread to areas adjacent to the

[2] These operations were performed on the monkeys by Dr. Edmund Applebaum, on the dogs by one of the authors (W. L.) and on human beings by Dr. Daniel Ziskin of this school.

stain pocket, a condition which might have occurred if the specimen had been prepared in aqueous solution. The first photographs were made by reflected light. Later the teeth were ground to a thickness of about 300 micra, mounted in canada balsam on slides and cover glasses and photographed again; this time with transmitted light.

RESULTS OF OBSERVATIONS OF DYE PENETRATION IN VITAL TEETH

The diffusion of the dyes from the experimental cavity in vital teeth showed the same general phenomena in all cases, inasmuch as they appeared to follow an organized path, staining only certain areas. The tubules were the channels responsible for the distribution of the dyes in the dentin. The deepest stained areas were those in which the dentinal tubules communicated directly with the dye deposit (*fig. 13c*). A small group of tubules, immediately adjacent to the base of the dye deposit were also stained. Examination of the sections under high magnification showed that the tubules were stained more deeply than the basis substance of the dentin; the appearance of stain in the basis substance was due to staining the lateral branches of the tubules. The plane in which the section was cut, affected the picture of the distribution of the dye. Where the plane ran parallel to the course of the dentinal tubules (*figs. 4 and 5*) the boundary between the stained and the unstained areas is sharply defined. *Fig. 6* on the other hand, shows a more diffuse distribution of the stain, a condition probably resulting from the fact that the plane of the section passes somewhat obliquely across the course of the dentinal tubules.

Discussion of the diffusional channels in vital teeth: The stain diffusion in the vital teeth occurred in two directions from the dye deposit i.e. towards the dental pulp and towards the periphery of the crown (*figs. 2 and 4*). The illustrations show the variations in staining, obtained with different dyes in the teeth of dogs, monkeys and man. The enamel of monkeys stained less readily than that of dogs. A definite relationship existed between the various types of dyes used and the degree of penetration obtained. Potassium permanganate, eosin, methylene blue and silver nitrate penetrated the enamel well in the teeth of dogs while the congo-red and borax-carmine showed less penetration. In each case, the enamel was not stained homogeneously, for the dye appeared to follow specific channels i.e. the bands

of Schreger and the rod sheaths (*figs. 1, 2, 3, 7 and 9*). Teeth stained with silver nitrate, potassium permanganate and argyrol showed stain particles in the dentinal tubules. The particles were seen also in the enamel, located in the enamel rod sheaths (*fig. 2*). Some of the silver nitrate had penetrated to the pulp and been carried into the dentin at a point lower down on the lingual half of the tooth, staining an area of tubules not involved in the stain pocket. Similarly, in *fig. 6*, a tooth of a monkey, we note that the eosin from the dye deposit has penetrated to the pulp and from this area, it has been re-distributed to the dentin on the opposite wall of the pulp chamber, staining this slightly.

The observations of the animal experiments described in this paper indicate that one author's (CFB) designation of the dental fluid as *dental lymph* is warranted. This term was selected in order to denote its probable similarity to nutritional fluids of the body. It is believed that the term "lymph" alone might be misleading, as it would suggest that it is simply lymph penetrating the dentin from the blood vessels of the dentinal pulp. The term "lymph" is applied to other fluids present in the body, whose constituents are at variance with true body lymph. The perilymph and the endolymph, found in the middle ear are definitely different from body lymph; the former being similar to the spinal fluid. This has but few cells—only one or two to the cubic millimeter—and the presence of these are probably due to the needle puncture, necessary to withdraw this fluid from the spinal cord. In short, the term "lymph" when it is applied specifically need not be histologically similar in composition to body lymph. Further, the term "dental" is preferable to "dentinal" as this fluid does not appear to be confined to the dentin, but is shown by the present animal experiments to also permeate the enamel of young teeth.

Observation of teeth before sacrificing animal: A possible criticism might be made of these experiments: it might be claimed that the dye did not diffuse through the dentin and enamel while the animals were alive; or that the stain was rubbed over the surface of the teeth while preparing the section. This is refuted by the three following observations: 1—the stain can be noted near or on the surface of the tooth before its removal from the animal; 2—the boundary between

stained and unstained dentin is sharply defined as in *figs. 4 and 5*, a condition not possible to duplicate by rubbing the stain over the surface of a section during its preparation; 3—the dye can be noted in the depth of the section when this area is brought into sharp focus under high magnification of the microscope. This evidence seems sufficient to conclude that the dye diffused through the teeth of the experimental animals during life.

A peculiar condition was noted in the case of one tooth of a dog: it showed a stain distribution illustrated in the accompanying diagram (*fig. 10*). The only part of the tooth which was stained externally was a ring below the incisal tip. It appears that abrasion had resulted in the formation of secondary dentin which blocked the tubules leading to the tip of the tooth, thus preventing stain penetration to this area. The tubules, which were not blocked and which were in connection with the dye deposit, distributed the stain in the form of a ring around the tooth.

The presence of a fluid in the teeth of dogs and monkeys being indicated by the above experiments raises the question of its origin. There are two possibilities, it may originate either from: 1) the saliva, or 2) the dental pulp. If the *saliva* is considered the agency which dissolved the dye, then, the distribution of the stain would probably be like that shown in *fig. 13A*. The saliva would reach the dyed deposit (DD) from the surface of the tooth, along the course of the dentinal tubules, leaving the area "1" unstained. Then it would dissolve the dye and stain area "2" on its way to the dental pulp. This is not the condition noted in our specimen for *both* areas "1" and "2" are stained. However, fluid coming *from the pulp* along the dentinal tubules would first traverse area "2" (*fig. 13B*) leaving it unstained; then it would reach the dye deposit (DD) and distribute this to area "1" on its way to the tip of the tooth. We note that here, as in the hypothesis of the saliva, that one of the two areas is left unstained in either case. This observation might suggest that both the saliva and a fluid originating from the pulp is responsible for the distribution of the dye in the experimental teeth. Such a conception would suggest that the saliva penetrates to the dental pulp, a conception that does not seem feasible.

The following observations indicate that a return flow of dental

lymph occurs: 1—the presence of the dye in the pulp (*figs. 1, 2, 4 and 5*), 2—the removal of the dye from the dentin. It was observed that the teeth were stained more deeply with eosin in one day than when the dye was allowed to remain in the teeth for longer periods—one week or two weeks, (*figs. 8 and 9*). Previous evidence of a return flow of the dental lymph has been advanced by Blackberg and Berke (21) who observed a decalcification of the fully formed dentin in teeth of dogs under severe calcium-phosphorous disturbances. This observation indicates that mineral salts may be extracted from dentin. These observations are presented as evidence that there is an efferent as well as an afferrent flow of dental lymph in the dentin.

The path followed by the dye in the experimental teeth indicates that the channels in the dentin connect with those in the enamel. This is shown by the observation that stained enamel is found only over areas of stained dentin (*figs. 1, 2 and 3*). The presence of a lymph plexus at the dentino-enamel border as described by Fish (9) is indicated by the observation that the dye is distributed somewhat more widely in this area. Whether the return flow of dental lymph to the pulp occurs by way of a second set of dental tubules (9) or by way of the circumtubular space (22 and 23) is uncertain. Work concerning this question is in progress.

The fact that the dye penetrated but little across the axes of the dentinal tubules indicates that the so-called sheath of Neumann and the dentin matrix is not highly permeable. This observation applies only to vital teeth; pulpless teeth show a different type of dye diffusion.

Difference in permeability of dentin and enamel: Stain diffusion occurred in the dentin of all teeth, irrespective of the type of the dye used, thus indicating that the size of the dentinal tubules is greater than the molecule size of the dye. In the enamel, on the other hand, conditions are different. Here, stains with a large molecule such as congo-red, did not penetrate, while the smaller molecule of methylene blue and eosin passed through the enamel to the surfaces of the teeth in dogs. This indicates that the permeability of the channels in the enamel is lower than those in the dentin. The teeth of dogs were found to be more permeable than those of monkeys. Preliminary experiments on human teeth indicate that they are somewhat less

permeable than those of monkeys and that age is an important factor controlling this condition. To sum up this phase of the investigation: The fluid which permeates the dentin and enamel of fully formed teeth of young dogs and monkeys appears to originate from the pulp. Further, it seems that the dental lymph diffuses in two directions, centrifugally and centripetally, thus presenting the possibility of carrying materials to and from the dentin; in short, suggesting metabolic activity in teeth.

DIFFUSION OF DYE IN PULPLESS TEETH

The diffusion of stains in teeth from which the pulps were removed is indeed surprising and possibly may throw some light on the activity of the dental pulp in regulating the metabolic activity of the calcified dentinal tissue. It has been shown that the study of the diffusibility of stains in vital teeth has established the presence of an afferent and efferent flow of dental lymph. The stained areas are limited to those dentinal tubules which pick up and transport the dye particles, while the remaining dentin and enamel, not in such connection with the dye deposit show no stain whatever. The following observations are presented in evidence that the dental pulp is the significant organ linking the calcified dental tissues with the nutritional system of the body. This statement is based on the fact that remarkable differences in stain diffusion in vital and pulpless teeth were observed.

In order to obtain data on possible differences caused by the absence of the dental pulp, devitalized teeth were treated in the same manner as vital teeth. For this purpose, three groups of experiments were performed involving five techniques:

Group A (in vivo): The pulps of dogs' teeth were extirpated, the root canals reamed and dried. The canals were packed with gutta percha, the pulp chambers filled with a dry dye and the cavity in the crown sealed with oxy-phosphate cement. In one case (*fig. 11*) eosin was placed one week after pulp extirpation, in another eleven months were allowed to elapse before the insertion of the dye. *Group B (in vitro):* Recently extracted non-fixed teeth were stained in the manner used for vital teeth i.e. a cavity prepared from the labial surface was filled with a powdered dye, allowing eight days to elapse before preparing ground sections. One set (B-1) was placed in an incubator, while the other set (B-2) was kept at room temperature. Both sets of teeth were wrapped in gauze and moistened with normal salt solution, to prevent drying. *Group C (in vitro), C-1 and C-2:* The teeth in this group were treated in the same manner as those in Group B-1 and -2 with this exception—they were first fixed in 10% formalin. The dyes in these teeth were allowed to penetrate for the same length of time i.e. for eight days, before ground sections were made.

All sections made from these groups show essentially uniform phenomena entirely *different* from those observed in vital teeth. In the dentin, the stain penetrated throughout the entire body of the tooth. The basis substance was stained as well as the tubules. The stain was not limited to the tubules involved in the stain pocket but spread, in some cases, to the apex of the tooth, crossing the axes of the dentinal tubules. The enamel also showed uniform staining, being accentuated along the bands of Schreger. The entire enamel cap was stained in contradistinction to vital teeth, in which the stained enamel was limited to the area over the affected tubules. In most of the cases the entire cementum was stained and in one case (*fig. 11*), the stain had penetrated into the alveolar process as far as the adjacent tooth.

In Group A, the results of the first case (*fig. 11*) in which the recently devitalized tooth of a dog was stained with eosin, the penetration was so marked that not a single unstained area was visible before the tooth was sectioned. The enamel, dentin, and cementum were deeply stained. This experiment was repeated in the tooth of another dog which had been devitalized for eleven months. In this case the diffusion of the dye had the same characteristics but the penetration was far less intense, very little of the stain penetrating the enamel or root dentin. It was noted in all cases that the penetration of the stain was inversely proportional to the length of time that the tooth was devitalized before staining.

Group B showed a marked penetration. In these cases, the stain pocket was prepared in the same manner as in vital teeth. The stained area was not limited to the tubules involved in the stain pocket but as in Group A, the enamel, cementum, and the entire body of dentin was stained. The same observations were made in Group C but the staining was less intense. In all experiments, human teeth showed less penetration than dogs' teeth.

The fact that the diffusion of stains in teeth from which the pulps were removed was radically different from that noted in vital teeth, shows that a vital pulp exerts some obscure influence on the dental tissues. One effect of a vital pulp appears to be that of regulating the rate of diffusion of dental lymph. We conclude, therefore, that certain physical and chemical changes which occur in dentin and

enamel are dependent to a large extent on the dental pulp and the dental lymph. These observations show further that experiments performed on extracted teeth do not duplicate conditions active in vital teeth. Consequently a comprehension of the physiological activity of dentin, enamel, and cementum can be obtained only by experimentation on vital teeth.

AGE FACTOR AND TOOTH PERMEABILITY

One subject remains for discussion i.e. the variable permeability of the dental tissues. *Fig. 12* shows two first incisors of the same patient, age 37, treated as in *fig. 13C* by Dr. Ziskin. The left first incisor shows greater permeability than the right. Also the gentian violet diffused through the dentin more than the eosin. The lesser stained right first incisor was abraded more than the left one. In another series of two first incisors, one having proximal silicate fillings, the unfilled central showed greater permeability. It may be concluded that abrasion and caries definitely decrease the permeability of dentin due to changes in the tubules. We have observed previously that dental lymph is present in the dentin and enamel during the life of the dental pulp and further that their permeability is moderate during this time. The diffusion of stain is radically increased *immediately after* the removal of the dental pulp, so that the dental lymph dissolves and carries the dry dye, enclosed in the tooth, throughout all the calcified tissues. It is clear that as long as dental lymph is present, a high degree of permeability is maintained in the tooth. Histological examination of well matured teeth, on the other hand, shows a decomposition of the dental lymph, thus replacing the previously present fluid content of the channels with gas and dried remnant of lymph. These changes can be noted clearly in the dentin.

Dentin is a calcified connective tissue having a matrix, similar to that of bone. Passing through this are hundreds of thousands of delicate channels in a cubic millimeter of matrix. These channels 2 micra in diameter, the dentinal tubules, contain processes of the odontoblasts of the dental pulp. It is these processes, the dentinal fibrils which transport the dental lymph. The slow metamorphosis of the dental lymph contained in the dentinal tubules can be observed clearly under the microscope. In *fig. 14*, we note the beginning of

the formation of gas bubbles, separated by transparent areas filled with fluid; the normal, fluid-filled tubules can be seen as mere outlines. As decomposition of the lymph progresses, the ends of the gas bubbles, which previously were rounded (*fig. 14*) becomes irregular, indicating that the lymph residue has dried completely (*fig. 15*). It is clear that the permeability of such dentin has been reduced greatly; fluids can penetrate such a tissue no longer by way of the blocked tubules, but only through the dense, calcified connective tissues matrix.

The conception of one of the author's (CFB-23) concerning the low permeability of pulpless teeth must be modified to apply only to those which have been devitalized for some time. The material on which the original histological observations were made, consisted of old pulpless teeth. Their condition (gas in dentinal tubules) was construed as making them highly impermeable to fluids. This has now been demonstrated by the present observation, but the permeability of *recently* devitalized teeth has now been shown to be high, hence we conclude that the removal of the pulp increases the permeability status of the dental tissues radically, but it does this only *temporarily*. After a time, the diffusibility of fluids in pulpless teeth reduces slowly, until finally they become almost impermeable.

SUMMARY

The placing of *dry* dyes in vital teeth of dogs and man demonstrates the presence and diffusion of "dental lymph." Maturity, abrasion or caries reduces greatly the permeability of the dental tissues. The removal of the dental pulp *temporarily* increases the permeability of the dental tissues, indicating that this organ is active in regulating the diffusion of this fluid.

These observations suggest the presence of a physiological activity in the dental tissues.

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Fig. 1. Tooth of a dog stained in vivo with potassium permanganate (7 days). Section not cut parallel to course of dentinal tubules, as in *figs. 4 and 5*, hence staining appears diffuse. Enamel stain clearly emphasizes bands of Schreger.

Fig. 2. Tooth of dog stained in vivo with silver nitrate (28 days). Stain pocket, having penetrated only labial half of tooth, stained only labial dentin and enamel. Lingual dentin unaffected indicating dye diffused through specific channels, tubules, and not across their axes (*see figs. 6 and 8*). Under high magnification, particles of silver can be seen in enamel rod sheath, indicating its permeability.

Fig. 3. Tooth of dog stained in vivo with potassium permanganate and trichloracetic acid (5 days). Staining medium fluid. Dye penetration less than that noted in *fig. 1*, indicating that acid reduced permeability of channels.

Figs. 4–5. Teeth of monkey stained in vivo with eosin (14 days). Sections were cut parallel to the long axes of the dentinal tubules showing clear line of demarcation between stained and unstained areas. Agfa Color.

Fig. 6. Tooth of a monkey stained in vivo with eosin (14 days). Diffuse distribution of stain probably due to fact that angle of section was oblique to course of dentinal tubules. Secondary staining of dentin present. Agfa Color.

Fig. 7. Tooth of monkey stained in vivo with methylene blue (14 days). This stain penetrated enamel more readily than eosin, staining it in various densities corresponding to bands of Schreger. Agfa Color.

Fig. 8. Tooth of dog stained in vivo with eosin (24 hours). Section, unfortunately, remote from long axis of tooth, hence only diffuse staining is seen. Agfa Color.

Fig. 9. Tooth of dog stained in vivo with eosin (14 days). Section cut at right angles to experimental cavity seen above pulp chamber. Stain more localized and less intense than section in *fig. 8*. Agfa Color.

Fig. 11. Pulpless tooth of dog. One week after pulp removal and filling canal, eosin was placed in pulp chamber for one week. Dye diffusion occurred throughout entire crown and root of tooth in spite of fact that the dye (D) was located only in crown. Agfa Color.

Fig. 12. Two first incisors of patient, age 37, each stained with eosin and gentian violet. Stain penetrated deeper in left tooth, indicating difference of permeability in teeth of same mouth. Dufaycolor.

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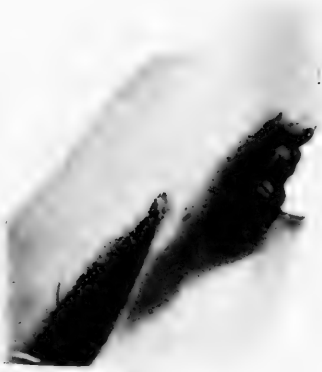

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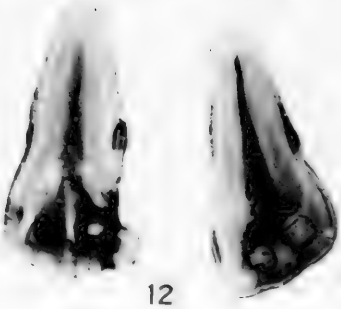

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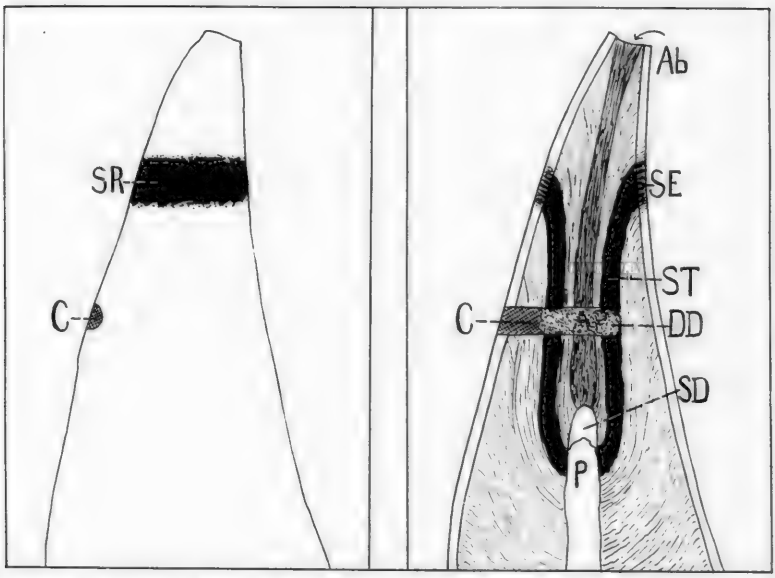

Fig. 10. Diagram of tooth of dog stained vitally. Distribution of dye in form of ring (SR), is explained by presence of secondary dentin (SD) resulting from abrasion (Ab); C—cement; DD—dry dye deposit; SD—stained tubules; SE—stained enamel; P—pulp.

Fig. 13. Three possibilities explaining manner in which dye deposit (DD) is dissolved and distributed through tooth of dog: A—Saliva (arrows) passes from without-inward to pulp, staining only area "2", B—Dental lymph diffusing to exterior from pulp (area "1"), C—Dental lymph diffusing in two directions. SE—stained enamel; SD—stained dentin; SSD—secondarily stained dentin from the pulp chamber. C illustrates technique of vital staining. Cavity drilled into labial surface and dye deposited in base of cavity (DD). Cavity then sealed with oxyphosphate cement.

Fig. 14. The formation of gas bubbles within the dentinal tubules illustrating an early stage in decomposition of dental lymph.

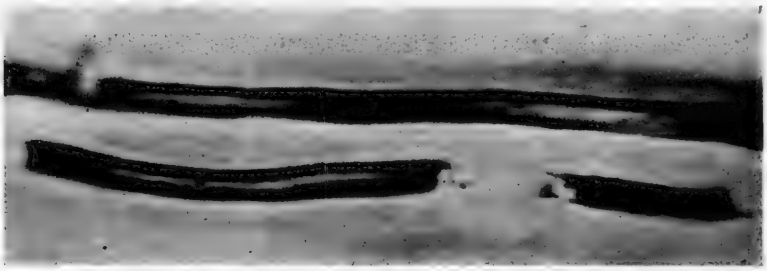

Fig. 15. Further decomposition of dental lymph. The rounded ends of the gas bubbles shown in *fig. 15* have become irregular, indicating a further drying of lymph.

ABSCESS PRODUCTION BY FUSIFORM BACILLI IN RABBITS AND MICE BY THE USE OF SCILLAREN B OR MUCIN

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On account of the difficulty of inducing lesions in animals by the use of pure cultures of fusiform bacilli, scillaren B and mucin have been used to lower their resistance.

The development of Plaut-Vincent infection in the mouths of dogs following intravenous injections of scillaren B observed by Wallace, Wallace and Robertson (1) suggested to Tunnicliff and Klein (2) the use of this drug together with pure cultures of fusiform bacilli in the production of abscesses in guinea pigs. Scillaren B is a crystalline glucoside isolated from the herb squill and has a cardiac effect similar to that of digitalis. Wallace, Wallace and Robertson suggested that the drug disturbed the metabolic processes so as to lower the resistance of the buccal mucous membranes. The experiments of Tunnicliff and Klein indicated that it produced a leukopenia and dilated the blood vessels in the skin of guinea pigs. Abscesses were produced by avirulent fusiform bacilli injected into an area first prepared by the subcutaneous injection of small doses of this drug until the guinea pig became slightly sick. Although only short, straight fusiform bacilli were injected, both bacilli and spirilla-like organisms were demonstrated in smears and sections of abscesses. Fusiform bacilli were recovered in pure culture from these abscesses and generally appeared as very fine, straight, bacillary or, sometimes, filamentous forms. In one culture from pus showing many spirilla-like organisms, fine granular spirilla-like forms mixed with bacilli and filaments were observed. The strain used in these experiments showed only straight forms during the first days of growth but, as a rule, spiral forms appeared later, indicating it contained the R factor. It has been

shown (3) that colonies of fusiform bacilli containing the bacillary forms (*fig. 1*) are smooth (*fig. 2*), while filaments and spirilla-like organisms (*fig. 3*) are associated with its rough colony (*fig. 4*).

Rabbits: scillaren B treatment—We have made similar experiments in rabbits and mice, using a strain of Gram negative, non-motile fusiform bacillus isolated three months previously from a patient with ulceromembranous gingivitis. At this time it produced small abscesses about 5 mm. in diameter when injected into the normal skin of shaved rabbits and was non-virulent when injected intraperitoneally into mice. The organism was grown anaerobically on either ascites phosphate or ascites plain agar, generally from 24 to 48 hours. The growth, usually from 1 or 2 slants, was suspended in salt solution before injection. This suspension of bacteria was injected into the shaved skin of three rabbits, one dose into a control area, another into a spot injected 4 to 7 times with 0.2–0.6 cc. of a scillaren B solution. One cc. of the scillaren B solution contained 0.0005 gms. of scillaren B. The drug produced large, indurated, sometimes hemorrhagic masses but did not make the rabbits sick. Fusiform bacilli and spirilla were not observed in the mouths of rabbits during the experiment.

Within 24 to 72 hours after the injection of fusiform bacilli, abscesses 0.5 to 2 cm. in diameter developed. Those in control spots were less than half the size of those in areas injected with scillaren B. Hemorrhages were sometimes observed on the abscesses. The rabbits were killed with ether, smears made and the abscesses removed. The tissue was fixed in formalin, embedded in paraffin, sectioned and stained by both the Levaditi method and with hematoxylin-eosin. Sections stained with silver showed fusiform bacilli and spirilla-like organisms when present. The sections stained with hematoxylin-eosin showed injected vessels, masses of polymorphonuclear leukocytes and eosinophils. Endothelial cells were seen, especially in the older abscesses.

One rabbit received 4 doses of scillaren B solution followed by the 24 hour growth of fusiform bacilli on ascites phosphate agar containing only short, bacillary forms (*fig. 1*). Abscesses developed in 48 hours, that in the scillaren B injected area being larger than that in the control spot. Four days after receiving bacteria the animal was

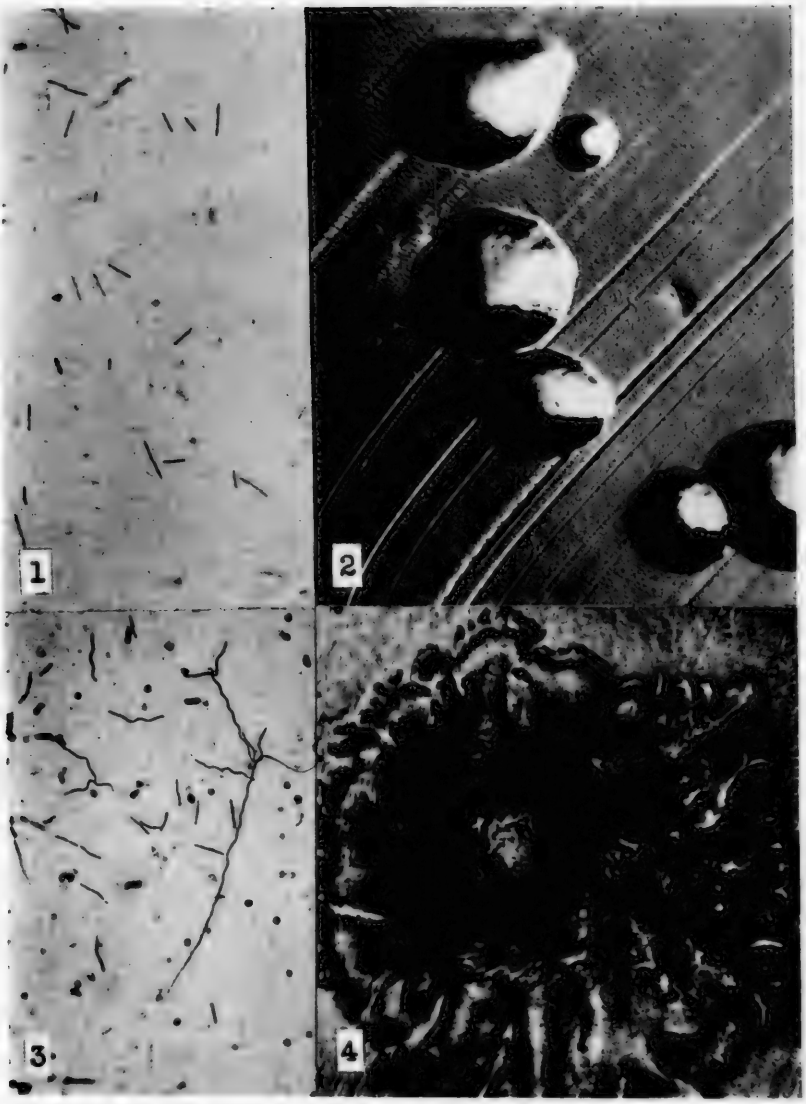


FIG. 1. Fusiform bacilli from smooth colony; Gentian-violet, ×1200.

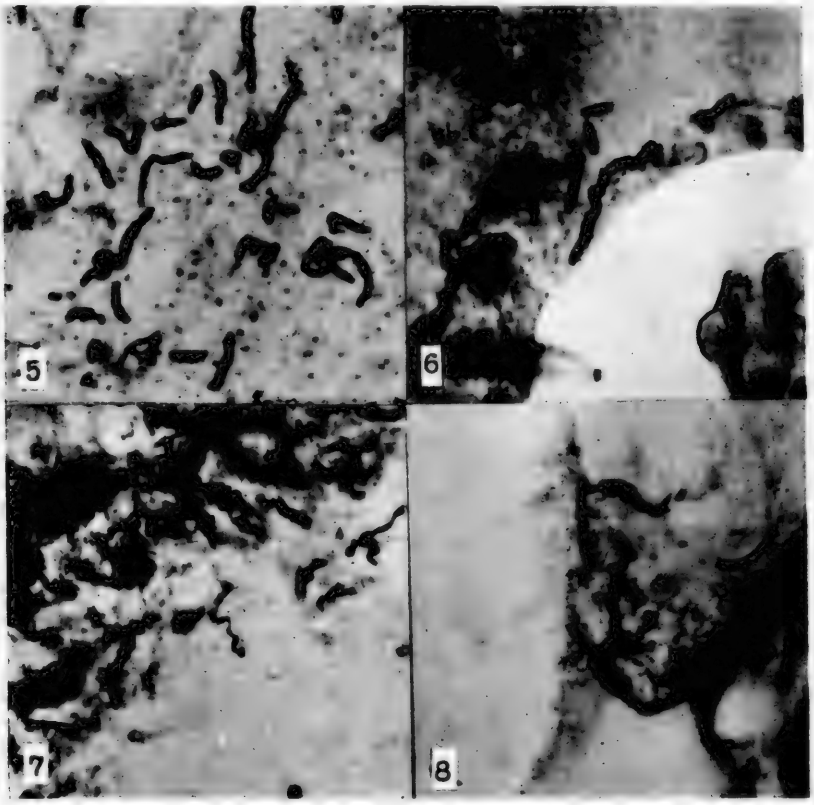
FIG. 2. Smooth colonies of fusiform bacilli; ×30.

FIG. 3. Fusiform bacilli and spirilla-like organisms from rough colony; Gentian-violet, ×1200.

FIG. 4. Rough colony of fusiform bacilli; ×30.

FIGS. 2 and 4 are reproduced from "Relation of Spiral Organisms to Rough Colony of Bacterium Fusiformis." Ruth Tunnicliff. J. Infect. Dis., 53: 280, 1933.

killed and sections of the abscesses were stained and studied. Both abscesses showed many straight and spirilla-like forms along the needle track and the surrounding tissues but none at the edge of the



FIGS. 5 AND 6. Fusiform bacilli and spirilla-like organisms from rabbit abscess; Levaditi stain, ×1600.

FIG. 7. Spirilla in section of ulceromembranous gingivitis; Levaditi stain, ×1600.

FIG. 8. Spirilla in section of gangrene of the face; Levaditi stain, ×1600.

abscess. In the abscess in the control area the spiral forms appeared to invade the tissue further than the straight forms. Spirilla-like forms were especially noticeable in the connective tissue. The spirilla-like organisms (*figs. 5 and 6*) in these sections resemble spirilla found

in sections from gangrene of the face (*fig. 8*) and ulceromembranous gingivitis (*fig. 7*) from which this strain of fusiform bacilli was isolated.

Another rabbit, receiving 7 daily doses of from 0.1 to 0.3 cc. of scillaren B solution, was injected with a suspension of bacilli grown on ascites phosphate agar. An abscess 0.5 cm. in diameter resulted in the control area. The abscess in the scillaren B prepared spot was 3 cm. in diameter and ulcerated 4 days after injection of bacteria The pus near the surface was thick and contained poorly stained fusiform bacilli, polymorphonuclear leukocytes (some of which contained bacilli) and many endothelial cells. Pus deep in the abscess removed with a sterile pipete showed a few short, rather thick, fusiform bacilli and many spirilla-like organisms, some in clumps. The spiral organisms were of two types, slightly wavy with irregular curves (*figs. 9 and 10*) and more tightly coiled forms (*fig. 11*). On account of the spirochetal appearance of the spiral organisms in the smear preparation, smears from the floor of the cage and the skin, mouth and intestine of the rabbit were examined but no similar spiral organisms were observed. Sections through the surface of this abscess showed masses of fusiform bacilli in more or less palisade arrangement around structureless material. Short, filamentous and a few tightly coiled forms were seen in the abscess along the path of infection. The organisms in the unprepared spot were mostly straight, bacillary forms.

The third rabbit received 3 injections of 0.2 cc. of scillaren B followed by fusiform bacilli grown on ascites plain agar. The abscess produced in the control area was one half the size of that in the prepared spot (2 cm. in diameter). Sections of the abscess in the prepared site showed long filaments and fusiform bacilli in the muscle (*fig. 12*). Associated with bacilli were short curved and spirilla-like organisms (*figs. 13 and 14*) mostly in the center of the abscess. A few spirilla-like forms were observed in muscle at the edge of the abscess.

Since ulceration was observed only once on the surface of an abscess produced in a rabbit by fusiform bacilli with the aid of scillaren B, we tried to produce necrosis by the injection of a culture or its filtrate into the blood 24 hours after the animal had received intradermal injections of culture or filtrate into the shaved skin. The filtrate was prepared by growing fusiform bacilli 48 hours anaerobically in ascites phosphate broth and filtering this culture through a Berkefeld N

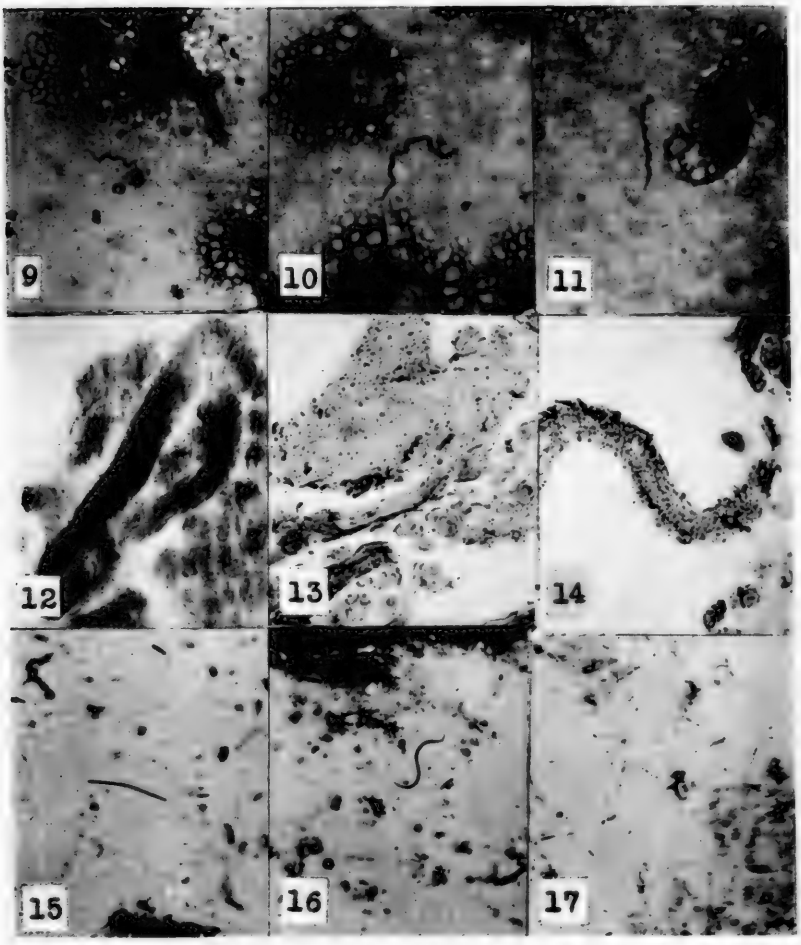


FIG. 9. Spiral form in smear from ulcerating rabbit abscess; Gentian-violet, ×1200.

FIG. 10. Same abscess; ×1600.

FIG. 11. Same abscess; ×1600.

FIG. 12. Section of rabbit abscess showing fusiform bacilli in muscle; Levaditi stain, ×1200.

FIG. 13. Section of same abscess showing fusiform bacillus and spirilla-like organisms; Levaditi stain, ×800.

FIG. 14. Section of same abscess showing clump of spirilla-like organisms; Levaditi stain, ×800.

FIG. 15. Filament and short fusiform bacilli in smear from mouse abscess; Gentian-violet, ×1200.

FIG. 16. Spirilla-like organism with fine projections in smear from mouse abscess; Gentian-violet, ×1200.

FIG. 17. Y form with projection in smear from mouse abscess; Gentian-violet, ×1200.

filter. This broth culture, or more often the twenty-four to forty-eight hour anaerobic culture on sheep blood agar slants, suspended in salt solution was used for the bacterial suspensions. Two guinea pigs were injected with 0.2 cc. of filtrate and of culture in different areas and 24 hours later one was injected in the heart with 1 cc. of filtrate, the other with the same amount of culture. Both guinea pigs became sick and tiny abscesses without necrosis resulted 24 hours later in the sites receiving bacteria followed by injection of bacteria into the heart. No abscesses occurred in the spots receiving filtrate into the skin followed by filtrate into the heart. Experiments in two other guinea pigs injected with bacteria both into the skin and heart were negative.

Small abscesses without necrosis were produced in four rabbits receiving bacteria intradermally. Two rabbits were injected in one area, one rabbit in two sites and one in five. All rabbits were given an intravenous injection of bacilli 24 hours later. The abscess of one animal that died after the injections was surrounded by petechiae. Sections of this abscess showed long filaments, a few of which were twisted.

An abscess 3 cm. in diameter produced by the injection of fusiform bacilli into a site prepared by 3 doses of scillaren B (0.2, 0.5 and 0.5 cc.) was not made necrotic by the intravenous injection of fusiform bacilli 24 hours later. Two rabbits, one injected in one spot with 0.25 cc. of filtrate, the other with the same amount of culture, followed in each case by 1–2 cc. of culture intravenously 24 hours later showed no reaction.

By the repeated intravenous injection of scillaren B an attempt was made to produce ulceromembranous lesions in the mouth of a rabbit, such as were observed in dogs by Wallace, Wallace and Robertson (1). Scillaren B solution was injected in 9 daily doses varying from 0.1–0.4 cc. Unlike dogs, the rabbit remained well during the experiment and no lesions were seen in the mouth during 2 weeks observation. This may have been due to the fact that the rabbit used was not susceptible to scillaren B and that neither fusiform bacilli not spirilla were found in the mouth before or during the experiment. After receiving scillaren B, the rabbit was injected in the upper lip with the growth of a blood agar slant suspended in salt solution.

The following day the face was swollen and there was much congestion around the site of injection. This disappeared in 2 days without producing any ulceration. No fusiform bacilli could be demonstrated in the mouth 2 days after injection.

Mice: scillaren B treatment—A control mouse injected intraperitoneally with 1 cc. of a salt solution suspension of fusiform bacilli remained well. Of three mice injected intracutaneously with 0.1 cc. of a scillaren B solution followed 24 hours later by the intraperitoneal injection of bacilli, one died in 3 days and the other two were made very sick, one 5 and the other 11 days after receiving bacteria. They showed injection of the vessels in the peritoneal cavity and a sticky peritonitis. Smears showed phagocytosis of fusiform bacilli, fusiform bacilli outside leukocytes, filaments and a few spirilla-like organisms. A mouse injected intraperitoneally with the fusiform bacillus recovered from the mouse that died was made very sick 3 weeks later and was killed. Peritonitis was present and smears showed a few fusiform bacilli.

Two mice injected into the skin with from 3 to 6 doses of 0.1 cc. of scillaren B solution followed by long fusiform bacilli developed sticky abscesses from 0.5–1 cm. in diameter. They contained, generally, large numbers of short fusiform bacilli, an occasional bacillus showing fine, curved ends, filaments, a few short curved forms and a few with fine projections (*fig. 16*). Fusiform bacilli with fine straight or wavy ends are seen in R cultures of fusiform bacilli. The same culture injected into a control area in another mouse did not produce an abscess.

When 1 cc. of fusiform bacilli and 0.1 cc. scillaren B were mixed and injected into the peritoneal cavity of a mouse, a mucoid abscess developed 8 days later showing bacilli, filaments and a few coiled forms.

The blood of mice treated as described was examined for spirilla in smears and by dark field illumination but none were demonstrated.

Scillaren B appears to lower the resistance of mice so that injection of non-virulent fusiform bacilli into the skin or peritoneal cavity is followed by abscess formation or peritonitis.

Mice: mucin treatment—After 9 months isolation this strain of fusiform bacillus generally showed bacilli with granules, filaments

and spiral forms characteristic of R cultures. A few experiments were made suspending fusiform bacilli in gastric mucin to favor infection as described by Nungester, Wolf and Jourdonais (4) to whom we are indebted for the mucin. Fusiform bacilli grown anaerobically on sheep blood agar were suspended in 1 cc. of either salt solution or mucin and injected intraperitoneally into two mice, neither of which became sick. Injections were then made into the skin. While no reaction was observed in four mice, one receiving only mucin and three receiving fusiform bacilli suspended in salt solution, mucoid abscesses varying from 15 mm. to 2 cm. in diameter were produced in from 1 to 4 days in ten of eleven mice receiving bacilli suspended in mucin. When the abscess appeared to be at its height from the third to the eighteenth day, the mouse was killed with ether. Although some showed little swelling on the surface, extensive abscesses were found under the skin. Fusiform bacilli containing granules were seen outside and within polymorphonuclear leukocytes. Filaments, sometimes in large numbers, and one Y form (*fig. 17*) were seen also. Few spirilla-like forms were seen although cultures containing them were frequently injected. Fusiform bacilli isolated from these abscesses and suspended in mucin produced abscesses in mice, but although passed through four mice did not become virulent and did not cause ulceration. The pus from one of these abscesses mixed with 1 cc. of mucin and injected into the skin of a guinea pig did not produce an abscess.

A guinea pig was injected into the shaved skin with fusiform bacilli suspended in salt solution in one spot and bacilli suspended in mucin in another. In 4 days a tiny abscess 1 mm. in diameter appeared in the area receiving bacilli in salt solution and an abscess 15 mm. in diameter at the site of injection with bacilli in mucin. This latter abscess persisted 2 weeks and showed large numbers of bacilli but only a few filaments and spirilla-like organisms. This experiment suggests that in guinea pigs an abscess produced by fusiform bacilli in mucin shows fewer spiral forms but persists longer than those resulting from fusiform bacilli in salt solution injected into sites prepared with scillaren B. Abscesses produced in this manner were present for not more than four days.

CONCLUSIONS

A fusiform bacillus isolated three months previously from a patient with ulceromembranous gingivitis produced abscesses about 0.5 cm. in diameter when injected subcutaneously into rabbits and abscesses from 1.0 to 2.0 cm. in diameter when injected into the skin, prepared by injections of 0.1 cc. of scillaren B solution. Although cultures showed only bacillary forms, spirilla-like organisms were demonstrated in smears and sections of the abscesses. Ulceration developed in one scillaren B prepared abscess in which typical spirochetal forms were seen.

This strain of fusiform bacillus nine months after isolation did not produce abscesses in skin or peritonitis in unprepared mice; but abscesses or peritonitis resulted if the mouse had previously received scillaren B intradermally or if this drug was mixed with the culture and injected intraperitoneally.

Mice injected into the skin or peritoneal cavity with fusiform bacilli suspended in mucin developed abscesses or peritonitis but no lesion resulted from the injection of mucin or bacilli alone.

More filaments and fewer spirilla-like organisms were demonstrated in lesions in mice than in rabbits although the culture injected was often rough and contained spiral forms.

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ACIDOGENIC AND ACIDURIC BACTERIA OF THE MOUTH AND THEIR POSSIBLE RELATION TO DENTAL CARIES

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The present investigation was undertaken with the hope of throwing some additional light on the etiology of caries from the bacteriological viewpoint. A quantitative study was made of the incidence of acidogenic and aciduric bacteria in saliva, at the site of initial enamel decalcification, and in advanced decay.[2]

MATERIALS AND METHODS

Three different media were employed—

1. The medium of Weiss and Rettger (1934).

Tomato juice	200 cc.
Peptonized milk (Difco)	10 gr.
Yeast extract (Difco)	5 gr.
Peptone (Difco)	5 gr.
Distilled water	800 cc.

The medium is adjusted to pH 8.0, with cresol red as indicator, before autoclaving. The final pH after sterilization should be 6.8–7.0. The tomato juice is obtained by overnight filtration of canned whole tomatoes. Two per cent agar is added to obtain a solid medium. To this basal medium were added 10 per cent of a 0.2 per cent solution of brom cresol purple prepared according to Clark (1928), and sterile hog serum (10 per cent) immediately before pouring the plates. The indicator was found to have no inhibitory effect upon stock strains of oral and intestinal lactobacilli, or upon several stock strains of streptococci.

Organisms which reduced the pH to 5.0 or below produced colonies which had a distinct yellow halo around them and stood out clearly against the intensely purple background. Precipitation of the serum protein in the medium by the acid formed caused a pronounced opacity in the halo.

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[1] This paper covers in part the dissertation submitted to the Graduate School of Yale University by the senior author as part requirement for the Degree of Doctor of Philosophy.

[2] A brief historical resumé and complete tabulated information will be found in the original dissertation in the Yale Sterling Library.

2. Modified Kulp medium (1927)—

Tomato juice	400 cc.
Peptone (Difco)	5 gms.
Peptonized milk (Difco)	10 gms.
Distilled water	600 cc.
Agar	20 gms.

The medium was adjusted to pH 5.0[3] before sterilization. The reaction was found to remain practically constant during twelve minutes' autoclaving at fifteen pounds extra pressure. Hadley (1933) used a similar medium in her quantitative determinations of lactobacilli in saliva, assuming that the low H-ion concentration (pH 5.0, according to her) was inhibitory to other organisms. We have observed, however, that some streptococci and yeasts are able to grow well on this medium.

3. Blood agar. In the later part of this study blood agar plates were used. These were prepared by adding 5 per cent sterile defibrinated whole-blood to the unmodified Weiss agar medium at a temperature of 45°C., immediately before pouring the plates.

The three media proved to be particularly favorable for the growth of the lactobacilli, at the same time permitting other mouth organisms to develop.

The following methods were used in collecting material for examination.

Saliva.—This was obtained by: (1) Chewing wax and depositing the saliva in a volumetric receptable, and (2) "washing" the mouth and teeth surfaces with sterile water, and collecting the washings. The first procedure was similar to that used by Rodriguez (1931), Hadley (1933) and other investigators.

Tooth scrapings.—These were obtained on sterile glass scalpels, by the method of Kligler (1915). The scalpels were made of 5 mm. pyrex glass tubing cut into three inch lengths. These were converted, as described by Kligler, into hoe-shaped scalpels having sharp cutting edges. This instrument is durable and well-suited for scraping the teeth surfaces. The straight end was wound in cotton and the scalpel inserted in a small vial for sterilization, following which the vials were placed in a desiccator over calcium chloride and dried to constant weight. It was essential to keep the scraper dry before use, since material adhering to the tooth surface at the site of initial decay is represented in milligrams. After the weight of the material was determined, the bent end of the scalpel was broken off and placed in a small sterile bottle containing a measured amount of water and 10 to 15 glass beads. Shaking of the bottle soon dislodged the scrapings and provided an initial dilution for plating.

Decay material.—This was obtained from freshly extracted teeth, which came as a rule from the same individuals who had furnished scrapings and saliva. External contamination was reduced to a minimum by placing the teeth immediately in a sterile test tube, and by cleansing the outer surface of the teeth with dilute alcohol before removing the decay material. The procedure used in making dilutions of this material was like that employed by Weinzirl and Newton (1914) in meat analysis.

In the plating of test material, 0.1 cc. of the desired dilutions was evenly spread over the surface of the solidified agar, in duplicate or triplicate. The original washings or suspensions were diluted 10 times and the results recorded on this basis. The dilution factor was apparently overlooked by Rodriguez (1930), in making his calculations.

All plates were incubated at 37.5°C. for 72 hours in closed jars containing ordinary air

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[3] Instead of 7.0, as was done by Kulp.

to which approximately 10% carbon dioxide by volume was added from cylinders of the pure gas. The plates were examined and counted at the end of 48 and 72 hours. Rodriguez (1931) incubated his plates in Brown anaerobic jars under partial anaerobic conditions and in the presence of 10% added carbon dioxide.

In the examination of the plates special emphasis was placed on organisms which were definitely acidogenic and aciduric. Acid-producing organisms produced, in the brom cresol purple agar medium, characteristic colonies which appeared conspicuous on the purple medium by their yellowish (acid) color and by a colorless opaque zone or halo around them. Non-acidogenic colonies were faintly purple, and were without the reduced zone. The number of acid-producing colonies was recorded. At the same time smears were made from 40 to 50 of such colonies and examined microscopically. Those which exhibited in smear preparations short or long, non-spore-forming, Gram-positive rods (single or in chains) were considered as lactobacilli. The streptococci were readily differentiated by their morphology. By this method the incidence of streptococci, lactobacilli and other acid-producing types was determined for each test sample.

The procedure followed with the "pH 5 agar" plates was similar in every way to the one employed for the "B.C.P." medium; all organisms developing on this medium were considered to be at least moderately aciduric.

On the blood agar plates the colonies were differentiated according to the degree of hemolysis. They were recorded as alpha (viridans type), beta (true hemolytic), and nonhemolytic. Smear examinations of from 40 to 50 colonies per plate were made.

The yeast, lactobacillus, and aciduric streptococcus incidence was tabulated. Types not falling into one of these 3 categories were placed under the heading, "other types." This mixed group included Gram-negative and Gram-positive cocci, Gram-negative bacilli, spore-forming bacilli, diphtheroid, lanceolate and oidium-like forms.

EXPERIMENTS AND OBSERVATIONS

Saliva: In a preliminary experiment saliva was obtained from 5 individuals. No history of the oral condition was solicited, and no tabulated record of the observations was made. The results are stated briefly here. There was a high incidence of Gram-positive acid-producing streptococci, Gram-negative cocci, Gram-negative bacilli, staphylococci, diphtheroids and yeasts, as was indicated by the plate colonies and individual slide mounts. No true lactobacillus-like forms were noted.

The first systematic study was conducted on 7 individuals. The saliva was collected about 2 hours after breakfast and the platings were made with the brom cresol purple medium. The results are given in Table I. The absence of lactobacilli and the comparatively high incidence of acid-producing streptococci are noteworthy. The individuals were all subject to caries. However, information as to whether decay was active at the time of the experiment was not

available, although all of the individuals had consulted a dentist within a year.

Several experiments were conducted with test material procured from patients attending the dental clinic in the New Haven Hospital, and full case records were obtained from the dentists conducting the clinic. Oral material was collected by the rinsing method described earlier in this paper. Brom cresol purple agar (pH 6.8–7.0) and acid agar (pH 5.0) plates were streaked with the diluted washings. The oral condition of the subjects varied from so-called "clean" to the extensive carious and "bad-mouth" condition, and the number of decayed teeth from 1 to 10 per patient. The decay was diagnosed as "arrested" or "progressive." Results obtained with material streaked

TABLE I

Results of examination of saliva of 7 individuals

CASE	NO. OF ORGANISMS PER CC. IN MILLIONS			
	Total	Streptococci	Lactobacilli	Other Types
I	4,080,000	750,000	None	3,330,000
II	3,340,000	300,000	"	3,040,000
III	7,530,000	420,000	"	7,110,000
IV	3,250,000	500,000	"	2,750,000
V	1,950,000	160,000	"	1,790,000
VI	1,610,000	510,000	"	1,100,000
VII	(overgrown with subtilis-like type)			

on the "B.C.P." agar medium from 18 patients are given in Table II' and with material applied on the "pH 5" agar plates from 14 patients in Table III. Surprisingly few lactobacilli were observed in the mouth washings. Only 2 of the 32 patients were lactobacillus-positive by the 2 methods used. These individuals exhibited caries to a marked degree. On the other hand, 15 of the patients presented a high streptococcus incidence. Also, in 9 instances yeasts were present in large numbers. The relatively high yeast incidence is of considerable interest. Little reference has been made in the literature to oral yeasts. In this investigation the presence of yeast forms was, with one exception, correlated with so-called progressive decay. Unfortunately, a thorough study of the relation of oral yeasts to caries could not be carried out in this study. Yeasts are known to be highly

TABLE II

Results obtained with saliva plated on brom cresol purple agar medium

CASE NO.	AGE	DIAGNOSIS* (CARIES)	NO. OF CARIOUS LESIONS	NO. OF ORGANISMS PER CC. IN MILLIONS				
				Total	Acidogenic Streptococci	Lacto-bacilli	Yeasts	Other Types
1	8	Progressive	9	9.4	0.3	0.22	0	8.88
2	25	"	5	5.5	0.5	0	0	5.0
3	17	"	6	9.2	0.9	0	0	8.3
4	23	Arrested	1	23.5	5.0	0	0.1	18.4
5	45	"	2	37.3	3.0	0	0	34.3
6	12	Marked	X**	8.0	0.7	0.1	0	7.2
7	14	Arrested	2	5.0	0.8	0	0	4.2
8	29	Slight	X	1.0	0.4	0	0	0.6
9	30	Slight	2	1.7	No count	0	0	0
10	14	Moderate	6	0.12	0.03	0	0.02	0.07
11	47	Slight	1	1.5	0.25	0	0	1.34
12	12	Progressive	8	0.80	0.2	0	0.5	0.1
13	13	"	X	5.1	1.7	0	1.0	2.4
14	10	"	7	2.6	2.0	0	0.6	None
15	24	"	4	11.0	1.0	0	1.0	9.0
16	13	"	5	17.8	5.9	0	1.9	10.0
17	15	"	10	3.8	0.0	0	3.8	0
18	10	"	2	3.4	0.0	0	3.4	0

* The diagnosis is based on macroscopic observation.

** X—number of carious lesions not ascertained.

TABLE III

Results obtained with saliva plated on "pH 5.0" agar medium

CASE NO.	AGE	DIAGNOSIS* (CARIES)	NO. OF CARIOUS LESIONS	NO. OF ORGANISMS PER CC. IM MILLIONS				
				Total	Strepto-cocci	Lacto-bacilli	Yeasts	Other Types
4	23	Arrested	1	7.2***				
5	45	"	2	26.5	2.1	0	0	24.4
6	12	Marked	X	4.1	0.30	0	0	3.8
7	14	Arrested	2	3.0	0.60	0	0	2.4
8	29	Slight	X	3.	1.2	0	0	1.8
10	14	Moderate	6	0.11	.006	0	0	0.104
11	47	Slight	1	0.30	0.1	0	0	0.20
12	12	Progressive	8	0.40	0	0	0.4	0
13	13	"	X	0.98	0.88	0	0.10	0
14	10	"	7	1.8	0.50	0	0.30	1.0
15	24	"	4	0**	0	0	0	0
16	13	"	5	3.4	2.8	0	0.60	0
17	15	"	10	0**	0	0	0	0
18	10	"	2	0**	0	0	0	0

* The diagnosis is based on macroscopic observation.

** No growth on any of the plates.

*** No definite counts obtainable due to the crowded condition of the plates.

X—Number of carious lesions not ascertained.

saccharolytic, as a rule; this, with the probability of their producing other enzymatic action, suggests that they may play an important rôle in dental decay.

Among other types of microörganisms observed in the oral washings, cocci were the most numerous. All of the types produced some degree of fermentation, as was indicated by color change of the medium.

The findings reported here by us do not support the claims of certain previous workers concerning lactobacilli as etiological agents in the caries process. The results are in agreement, however, with the observations of Enright, Friesell and Trescher (1932) who reported finding 39 per cent of 85 patients exhibiting incipient caries apparently free from lactobacilli. Conversely, they showed that 52 per cent of 63 "caries free" patients were lactobacillus-positive. These workers state, "This evidence certainly does not indicate that lactobacilli are important factors in the etiology of caries of the enamel."

The high streptococcus incidence observed by us in oral washings is in harmony with the earlier reports of Goadby (1903), Kligler (1915), Hartzell and Henrici (1917), Clarke (1924), Maclean (1926) and others. In conformity with the demonstration of streptococci in large numbers in the mouth, their highly saccharolytic nature further stamps them as factors which invite most serious consideration in a study of the etiology of caries.

Tooth Decay Material: In an attempt to correlate the findings in the oral washings with the flora of decayed teeth, quantitative determinations were conducted on extracted teeth from 14 dental patients. The results are shown in Table IV. As was to be expected, the results were irregular. In 10 of the cases streptococci were demonstrated readily on the 2 types of media, (pH 5.0, and neutral brom cresol purple agar) and lactobacilli in 6. Gram-positive staphylococci were found in pure culture in one instance, and Gram-negative cocci in another. In 5 instances no growth was obtained in the plating of decay material. Negative findings are apparently not uncommon. Maclean (1926) reported such in 18 instances. De Vries (1930), in a study of layer material from 33 decayed teeth, reported an irregular streptococcus incidence in all cases.

The flora of the decayed tooth, especially in the secondary stage of caries, can bear at best only a remote relationship to the more im-

portant, initial breakdown. There are definite indications, however, of the streptococci playing a more important part in this advanced stage of caries than the lactobacilli. This may be attributed, not

TABLE IV

Results of examination of decay material from extracted teeth and of tooth scrapings

CASE NO.	AGE	NO. OF CARIOUS LESIONS	DIAGNOSIS (CARIES)	NUMBER OF ORGANISMS PER MILLIGRAM IN MILLIONS									
				Brom cresol purple medium					Acid ("pH 5.0") medium				
				Total	Acidogenic Streptococci	Lactobacilli	Yeasts	Other Types	Total	Streptococci	Lactobacilli	Yeasts	Other Types
A. Decay material													
2	25	5	P	3.0	0.8	1.5	0	0.70	0[1]	0	0	0	0
6	12	x	M	0.5	0	0.50	0	0	0.30	0	0.30	0	0
5	45	2	Ar	10.0	6.5	0	0.20	3.3	14.4	1.4	0	0.20	12.8
7	14	2	Ar	5.0*	0	0	0	0	0**	0	0	0	0
8	29	x	S	1.1	0.8	0.14	0	0.16	0**	0	0	0	0
9	30	2	S	0**	0	0	0	0	0[1]	0	0	0	0
10	14	6	M	0.055	0.011	0.023	0	0.021	0.066	0.006	0.02	0	0.04
12	12	8	P	5.5	1.13	0	0.04	4.33	2.76	1.54	0	0.05	1.17
13	13	x	P	3.19	0.29	0	0.36	2.54	0.17	0	0	0.17	0
14	10	7	P	0***	0	0	0	0	0***	0	0	0	0
15	24	4	P	5.0	4.96	0	0.04	0	0**	0	0	0	0
16	13	5	P	0**	0	0	0	0	8.7	8.0	0	0	0.70
17	15	10	P	5.0****	0	0	0	0	4.69	1.86	2.83	0	0
18	10	2	P	—	0.89	0	0	0	1.11	1.01	0.10	0	0
B. Tooth scrapings													
19	13	12	P	3.2	1.1	0	0.40	1.7	5.76	5.76	0	0	0
20	11	6	P	OV	OV	OV	OV	OV	32.7	30.	0	0	2.7
21	11	3	Ar	0**	0	0	0	0	0**	0	0	0	0
22	36	0	F	5.44	0.90	0	0.20	4.34	1.2	1.2	0	0	0
23	35	2	M	1.2	0.98	0.15	0.07	0	0.66	0	0.66	0	0
24	19	6	Ad	11.25	10.	0	0	1.25	0**	0	0	0	0

x = No. of carious lesions not determined.
* = Staphylococci.
** = No visible growth observed on any plates.
*** = Overgrown.
**** = G. negative cocci
OV = Overgrown with spreader.
0[1] = No determinations made.
P = Progressive.
Ar = Arrested.
F = "Free."
M = Moderate.
Ad = Advanced.
S = Slight.

TABLE IV—*Concluded*

CASE NO.	AGE	NO. OF CARIOUS LESIONS	DIAGNOSIS (CARIES)	NUMBER OF ORGANISMS PER MILLIGRAM IN MILLIONS									
				Brom cresol purple medium					Acid ("pH 5.0") medium				
				Total	Acidogenic Streptococci	Lactobacilli	Yeasts	Other Types	Total	Streptococci	Lactobacilli	Yeasts	Other Types
B. Tooth scrapings—*Continued*													
25	14	4	Ad	12.5	5.0	0	0	7.5	0.05	0.05	0	0	0
26	9	8	P	7.9	4.3	0	0	3.6	0**	0	0	0	0
27	18	1	S	6.24	5.16	0	0	1.08	0**	0	0	0	0
28	16	16	Ad	—	4.1	0	0	0	0**	0	0	0	0
29	41	6	Ad	1.2	1.2	0	0	0	0**	0	0	0	0
30	29	8	M	10.3	7.8	0	0	2.5	0**	0	0	0	0
31	8	8	P	6.12	4.44	0	0	1.68	0**	0	0	0	0
32	11	5	M	16.0	2.75	0	0	13.25	0.48	0	0	0.48	0
33	40	5	P	41.24	16.48	0	0.48	24.28	4.0	0.4	0	3.2	0.4
34	9	x	M	23.2	1.12	0	4.4	17.68	0**	0	0	0	0
35	12	2	S	4.56	3.9	0.57	0	0.09	12.63	6.0	0.09	0	6.54
36	12	3	S	4.0	3.72	0	0	0.28	0**	0	0	0	0
37	8	5	S	11.2	0.5	0	0	10.7	0**	0	0	0	0

only to the greater invasiveness of the streptococci, as was held by Hartzell and Henrici (1917), but also to the higher protein content of the dentine, thus providing a more favorable environment for their growth. We have observed in smears of layer material from decayed teeth an increasing streptococcus incidence in the direction of the deeper layers. Streptococci in the dentine may bear an important relationship to apical infection and abscess formation on the tooth.

Tooth Scrapings: It is generally conceded that caries is an external phenomenon originating from the progressive decalcification of the outer tooth enamel. Some investigators adhere to the theory of the decomposition of the interstitial columnar material. Histological sections of decayed teeth, however, do not support this hypothesis; they reveal that the inter-columnal material may still be intact, although the enamel presents a picture of disintegration. It is conceivable, therefore, that bacteria associated with initial decay are the organisms which are responsible for, or important factors in, this

condition. For this reason a series of observations was made on enamel scrapings.

Material was obtained from the teeth surfaces of 19 individuals attending the dental clinics. Efforts were made to procure teeth undergoing inital caries, as diagnosed by the usual ocular method. No scrapings were made from teeth that were in the so-called "second stage" of decay. The amount of test substance varied from 2 to 10 milligrams. The neutral brom cresol purple and the acid (pH 5) agar media were supplemented in 6 instances by the blood agar medium described earlier in this paper. The results of these findings are given in Tables IV, B and V. There was a high streptococcus

TABLE V

Results of examination of tooth scrapings (blood agar media)

CASE NO.	AGE	DIAGNOSIS	CARIOUS LESIONS	TYPES OF STREPTOCOCCI PER MG. IN MILLIONS			
				Alpha	Beta	Gamma	Total
32	11	Moderate	5	23.	2.5	2.0	27.5
33	40	Progressive	5	20.	0	.64*	20.
34	9	Moderate	x	0	0	16.**	0
35	12	Slight	2	0	0	2.25	2.25
36	12	"	3	2.6	1.6	6.5	10.7
37	8	"	5	.35	0	2.25	2.6

* = Yeast cells.

** = Gram-negative cocci.

incidence in all but two cases, as revealed by the B.C.P. medium. In only 3 instances were lactobacilli observed.

With the acid (pH 5) agar 11 of the 19 determinations gave negative results. This unexpected outcome of the tests may be attributed to the acidity of the medium. However, if viable aciduric lactobacilli had been present they should have experienced no serious difficulty in developing on this medium. In 6 instances streptococcus colonies were observed, once in pure culture, and on only 2 occasions were lactobacilli present, once in pure culture. The acid medium cannot be considered specific for the aciduric lactobacilli, since some streptococci and yeasts find it suitable for growth, as will be shown later. The necessity of making smear examinations in order to establish

the types of organisms growing on the plates is further established by these observations. Yeast-like organisms were observed in relatively large numbers in 6 instances on these 2 types of media. These findings do not support those of other workers in the field who used an acid broth enrichment procedure, and reported the presence of relatively high numbers of lactobacilli. Their results can be explained readily on the basis that in such a medium only a few viable lactobacillus cells present in the original inoculum may increase to an enormous number during the course of incubation. Such results have no quantitative significance, whatsoever.

The blood agar medium was used in 6 instances. In 4 of these it exhibited a high incidence of alpha hemolytic streptococci. Beta hemolytic streptococci were present in only 2 of the cases. On one occasion yeasts were observed, and in another Gram-negative cocci. Lactobacilli were not detected at any time. The results with the blood agar medium tend to support the findings with the other media in relation to the high incidence of streptococci on the surfaces of teeth subjected to initial decay.

During the course of these observations an opportunity presented itself for obtaining material from the teeth surfaces of 2 monkeys. Two observations were made on one animal (M1) and one on the other (M2). The salivary glands had been extirpated 2 years previously from M1. Both of the animals were "caries-free." The results obtained with the tooth scrapings from the 2 animals on 2 types of media (B.C.P. and "pH 5" agar) were of considerable interest, because they showed a high incidence of acidogenic organisms, including both streptococci and lactobacilli. The prevalence of strongly saccharolytic bacteria in the mouth of primates may be correlated with the high carbohydrate nature of their diet. Howett, Flemming and Simmonton (1928) observed an increase in the acidogenic organisms of the oral flora of men subsisting on a high carbohydrate diet. The high incidence of acidurics, coincident with the absence of caries, is of particular interest, although one is not justified in drawing definite conclusions from results obtained on such a small number of examinations.

CLASSIFICATION OF THE ISOLATED ACIDOGENIC AND ACIDURIC ORGANISMS

In an effort to clarify the taxonomic status of the oral lactobacilli and streptococci, cultural, biochemic and serologic studies were conducted on pure cultures isolated during the course of this investigation.

Oral Lactobacilli: The oral lactobacilli grow well on the surface of the agar plates, producing smooth, raised, white glistening colonies (Y type). The deep colonies in poured plates are compact and ovoid, some exhibiting a slight roughening or filament formation in the center. This type resembles more or less the "tam-o-shanter" colony of McIntosh, James and Lazarus-Barlow (1924), of their "*L. acidophilus-odontolyticus*." The colonies are in sharp contrast to the rough, X type colonies of intestinal *L. acidophilus*. Morishita's (1928) observation that the oral lactobacilli develop on the surface of agar at temperatures as low as 12° to 14°C. have been substantiated repeatedly in this laboratory. The intestinal strains, as has been shown again and again, do not produce visible growth at 20°C. or below. Enright, Friesell and Trescher (1932) reported that their lactobacillus strains which produced a rough (fuzzy) type of colony did not grow at 15°C. We have observed oral lactobacilli to develop as low as 10°C. On the other hand, we have never demonstrated visible growth of intestinal *L. acidophilus* below 20–22°C.

Due to observed irregularities in the ability of the lactobacilli to attack carbohydrates, differentiation on this basis has proven very unsatisfactory. Rahe (1918) considered glucose, maltose, lactose and sucrose as differential sugars for the classification of *L. acidophilus*. Bunting and Palmerlee (1925) classified their "*B. acidophilus*" strains according to Rahe's scheme. In this investigation biochemical observations were conducted with 15 strains of oral lactobacilli. The following 10 carbohydrates were used: sucrose, lactose, raffinose, dextrose, maltose, levulose, galactose, inulin, dextrin and starch. The 15 strains attacked all of the carbohydrates except inulin. According to these results, the fifteen strains should be placed in Group I (oral lactobacilli) of Enright, Friesell and Trescher (1932), who also included in the same group the "*B. acidophilus*" of Bunting and Palmerlee (1925) and the "*B. acidophilus odontolyticus*" of Rodriguez (1922).

Attempts have been made to classify the members of the *Lactobacillus* genus by serologic methods. Enright and his co-workers reported agglutination of the oral types by a homologous anti-serum in high dilution, but failure of the same serum to agglutinate the rough intestinal types. Morishita (1928) observed the absence of cross agglutination between oral and intestinal types, except in low dilutions. Serologic tests were conducted by us with anti-sera prepared against 2 oral strains of lactobacilli and 1 of intestinal origin. The anti-sera were prepared by injecting the organisms into the marginal ear vein of rabbits. Agglutination tests were conducted with these sera and 22 strains of oral lactobacilli and 3 of intestinal *L. acidophilus*. Only 4 of the 22 oral strains of lactobacilli were agglutinated by the anti-serum of the intestinal type. Three of these were in a dilution of 1–80 and one in a dilution of 1–640. Conversely, none of the oral anti-sera agglutinated the intestinal types. The oral anti-sera agglutinated the oral types in high dilution in all but 2 instances.

As a result of the cultural, biochemic and serologic observations made by us, and of the accumulation of evidence in this and other laboratories, we are led to the unqualified conclusion that the oral lactobacilli should be considered as a type entirely distinct from *L. acidophilus* of the human intestine.

Streptococci: A study was made of the strains of acidogenic and aciduric streptococci isolated in this investigation. For the preservation of stock strains it was found highly advantageous to use the egg-meat medium of Rettger (1906). Sixteen strains of streptococci isolated from saliva, tooth scrapings and tooth decay material were used in this work.

The streptococci ordinarily develop on agar media as small coccobacillary cells varying from 0.5 to 0.8 micron in breadth, and from 1.0 to 1.5 micron in length. Elongated club forms are often observed. The tendency toward long chain formation on solid media is slight. Irregular masses of cells generally appear. In broth, however, long chains of from 10 to 30 spherical or globoid cells predominate. They are distinguished readily from the lactobacilli by their morphology. The characteristic 48 hour streptococcus colonies are round, smooth, slightly raised, more or less glistening, opaque, and grayish white to

colorless on the acid (pH 5.0) agar. On the neutral brom cresol purple agar they assume an olive green to yellow color, due to the indicator. The colonies tend to adhere to the surface of the medium, and may be removed intact from the agar surface with the platinum needle.

Differentiation of the streptococci according to their biochemic activities has been attempted by Andrewes and Horder (1906), Holman (1916), Blake (1917) and Topley and Wilson (1929). The classifications were based on the utilization of mannitol, salicin, lactose and raffinose. In this investigation dextrose, maltose, dextrin, galactose, inulin and sucrose were included with the above-mentioned carbohydrates. The results obtained with the 16 strains of streptococci were irregular. It was apparent that biochemic methods afforded little in the way of streptococcus differentiation.

In an effort to determine the serologic relationship of the oral streptococci to each other and to the intestinal lactobacilli, anti-sera were derived with 3 strains of streptococci isolated from the mouth during this investigation. Agglutination antigens were prepared with 16 strains of oral streptococci, 2 of a known hemolytic streptococcus, and 3 intestinal strains of *L. acidophilus*. The method of Jennings (1926) was used in the preparation of the streptococcus antigens; this decreased to a marked degree the tendency of the streptococcal types to auto-agglutinate. The results of these observations displayed a marked serologic homogeneity of the oral streptococci isolated from different mouths. *L. acidophilus* (intestinal type) and the hemolytic streptococcus strain were not agglutinated by the oral streptococcal anti-sera.

Since there have been claims that the oral streptococcal types are but variants of the oral lactobacilli, cross-agglutination tests were conducted with the various anti-sera and strains mentioned in this report. The results are briefly summarized as follows: *L. acidophilus* (intestinal, X type) anti-sera did not agglutinate any of the oral streptococcus strains; 4 oral lactobacillus strains were agglutinated, 1 in a dilution as high as 1:640, and 3 in a dilution of 1:80. The oral lactobacillus anti-sera agglutinated 2 of the oral streptococcus strains in a dilution of 1:160; the others were negative. One dental lactobacillus strain was agglutinated by the oral streptococcus anti-sera in a dilution of 1:640, and slight agglutination of 8 other oral lacto-

bacillus strains was noted in low dilutions (1:40 and 1:80) by 1 of the 3 streptococcal anti-sera, the homologous titer of each of which was 1:1280. The results obtained do not support claims that the oral streptococci are variants of the oral lactobacilli.

The streptococci studied in this investigation do not possess the same cultural and biochemical characteristics as *Streptococcus salivarius* of Andrewes and Horder (1906) or *Streptococcus mutans* of Clarke (1924), and are, therefore, not specifically designated at this time as being either of these 2 types, although in several respects they resemble both.

EXPERIMENTAL PRODUCTION OF CARIES

There have been various claims of caries production *in vitro*. Miller (1903), using what must have been a mixed culture of oral bacteria and a bread pabulum, observed enamel decalcification in extracted teeth. Goadby (1903) reported similar results with *Staphylococcus viscosus* in broth. McIntosh, James and Lazarus-Barlow (1922) observed enamel dissolution of teeth placed in a broth culture of their "*L. acidophilus-odontolyticus*." Clarke (1924) and Maclean (1926) noted enamel decalcification in vitro by *Streptococcus mutans*. Bunting and Palmerlee (1925) employed an oral strain of "*B. acidophilus*" in broth and reported decalcification of the exposed areas of partially shellacked teeth kept in the broth culture. Negative results were obtained by them with a tooth that was exposed for 3 months to a culture of an oidium-like organism. It is apparent from this brief review that enamel decalcification can be induced apart from the mouth by several different oral bacteria.

The authors conducted a series of *in vitro* experiments on decalcification, using 1 strain of an oral streptococcus, 1 of an oral lactobacillus, and the 2 strains in combination. The method adopted was that used by Bunting, namely shellacking the teeth, but leaving an area or window for exposure to bacterial action. After sterilization the teeth were suspended in tubes of Weiss broth and the medium inoculated with 1 cc. of a 24 hour broth culture of the respective organisms. The teeth were transferred daily to tubes of fresh broth, new inoculum being carried over each time on the teeth. All tubes were incubated at 37°C. This procedure was carried out on each tooth for a period

of 10 weeks. Colorimetric hydrogen ion determinations were made of the tubes after each 24 hour incubation period. The pH was found never to be lower than 4.2.

Examination of the teeth at the end of the 10 weeks period revealed decalcification of the exposed enamel surface by both the streptococci and the lactobacilli, alone and in combination with each other. There was no marked difference in the degree of enamel dissolution brought about by the 2 organisms. The conclusion seems warranted, therefore, that the oral streptococci are as capable of producing enamel decalcification over long periods of time as are the oral lactobacilli.

GENERAL DISCUSSION

In a total of 65 individuals susceptible in varying degrees to dental decay, 12, (18.46 per cent), exhibited lactobacilli. The number of viable aciduric bacilli varied from 20 thousand to approximately 3 million lactobacilli per mg. of decay material from the extracted carious teeth. In scrapings from tooth enamel the lactobacillus incidence varied from 90 thousand per mg. in material from initial enamel decay to 5 million per mg. in scrapings from the teeth of monkeys which had never experienced caries. A wide range of variation is evident. The low percentage of lactobacillus positive cases, and the high lactobacillus findings in the mouths of "caries-free" primates do not support the specificity claimed by some for this organism in caries etiology. The media and methods used in our investigation were conducive in every instance to the growth of the lactobacilli, when these were present in viable form.

The occurrence of acidogenic streptococci in 55, (84.61 per cent), of the 65 cases examined should be highly significant, particularly in its bearing on the existing controversy regarding etiology. The streptococcus plate counts ranged from 6 thousand to 22 million cells per cc. of saliva, and from 50 thousand to 30 million per mg. of tooth scrapings. In view of these data, and of the highly acidogenic nature of these organisms, the oral streptococci should not be disregarded as a possible important factor in the etiology of caries. This statement is further supported by the observation that *in vitro* decalcification of enamel was brought about as readily by the oral streptococci as by the oral lactobacilli. Furthermore, the 2 types of organisms are distinct

entities; this was demonstrated conclusively by the cultural and serologic studies conducted by us. It was not, however, possible to place the oral streptococci under the type species *Streptococcus mutans* Clarke (1924) or *Streptococcus salivarius* of Andrewes and Horder (1906). What is of distinct importance also is that the oral lactobacilli are markedly different from the intestinal types (L. acidophilus and L. bifidus), particularly the commonly observed X and XY forms, on the basis of their cultural and serologic properties.

Throughout the investigation attention was given also to organisms other than mouth streptococci and lactobacilli. Gram-negative cocci and leptothrix- and yeast-like forms were often observed by us to be very abundant. If the claims made by some workers that strong H-ion concentration is not necessary for enamel breakdown, these organisms may assume a significant rôle in tooth decay. The possibility of their participation in the caries process should appear all the more plausible when we consider the pronounced buffering action of human saliva. With the high proportion of tri-calcium phosphate in saliva, and in view of the fact that enamel is composed almost entirely of tri-calcium phosphate (90 per cent), it is doubtful if a high acid concentration is ever reached on the tooth surfaces. Large numbers of organisms producing continuously small amounts of acid may be of as great importance as the highly acidogenic types. Finally, we cannot exclude the possibility of bacterial enzymes elaborated in the mouth as being a factor of no little importance in the march of dental decay, particularly after initial enamel injury.

CONCLUSIONS

If, as has been quite generally assumed, bacteria play a significant rôle in caries, through their biochemic products, mouth streptococci must be regarded as agents in the pathologic process, because of their frequent and abundant occurrence in the mouth, and on account of their decidedly acidogenic nature.

The oral streptococci are far removed from the lactobacilli in the biologic scale, and should at no time be confused with the latter.

The oral lactobacilli are of the "Y" (smooth, solid) type, and are distinct culturally and serologically from the now generally recognized "X" (rough) intestinal lactobacillus type.

Microörganisms other than the streptococci and lactobacilli occur in the oral cavity, and in sufficient numbers to be deserving of further bacteriologic study with respect to their possible relation to dental caries. Yeast and leptothrix forms in particular may prove to be of some significance.

In view of the marked differences noted between the various authentic strains of *L. acidophilus*, on the one hand, and the oral lactobacilli, on the other, and in order to avoid further confusion in the literature, the name *L. acidophilus* should not be applied to the mouth lactobacilli.

Acknowledgment is made here of the valuable aid given by Dr. Frank Kanthak, Dr. Sumter Arnim and Dr. Bert G. Anderson, in the collecting of the oral specimens and in the diagnosis of the oral condition of the patients from whom the material for study was obtained at the dental clinic.

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EFFECTS OF PREGNANCY, MOUTH ACIDITY, AND AGE ON DENTAL CARIES[1]

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The widespread idea that pregnancy is a cause of dental decay has been questioned. In the present study an attempt was made to throw further light on the problem. An index of caries based on the number of decayed tooth surfaces (Bodecker's Caries Index) was calculated for each of 324 pregnant women referred routinely from the Sloan Hospital for Women (*fig. 1*). Each patient received full mouth radiographs, allowances were made for missing teeth, and occlusal grooves large enough to admit the sharp point of an explorer were set down as cavities. The caries noted is of course an accumulation (see Bodecker's Caries Index) so that allowance must be made for age in comparing the observations on teeth with the number of past pregnancies. A further supposedly relevant variable is the hydrogen ion concentration of the saliva, which was recorded in each case because of the oft presumed relationship of caries to mouth acidity. In recording the salivary pH, unstimulated saliva was analyzed by the colorimetric method.

In addition to these pregnant women, 31 women who had never been pregnant were examined in the same way for comparison. The nonpregnant group consisted of a miscellany of hospital employees, patients coming in for reasons other than caries, et al. It is not possible to obtain a strictly comparable control of this nature, and the statistical results may perhaps be interpreted to mean that these 31 are not a random sample with respect to caries distribution, despite all precautions. Their mean caries index is substantially higher than that of the pregnant group, which may be interpreted to mean either that pregnancy prevents caries, or that the selection of cases

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was not random. But if we assume that this is a random group, the evidence agrees with that obtained from within the pregnant group by comparing these with differing numbers of pregnancies, in contradicting the traditional view, and suggesting rather definitely that pregnancy in some way tends to *prevent* tooth decay. It is clear at any rate that, in this sample, repeated pregnancy is in some way associated with some condition which prevents tooth decay, and to a significant extent.

Of the 324 pregnant cases, 164 were in their first pregnancies, one in her eighth, and the rest were intermediate. The average duration of pregnancy up to the time of examination was 5.76 months. Many ethnic groups were represented, the most numerous being the Hebrew.

By the method of least squares we fitted the regression formula

$$y = a + b_1x_1 + b_2x_2 + b_3x_3,$$

where: y = caries index, ranging from 4 to 94 for the various patients; x_1 = age in years (between 17 and 40 in all cases); x_2 = number of the current pregnancy (ranging from 1 to 8); x_3 = hydrogen ion concentration of saliva (6.30 to 7.10); and a, b_1, b_2, and b_3 are constants to be determined from the data; the primary interest of our problem is in the values, and particularly the signs, of the b's.

For the pregnant cases the means of the four variates and their correlations are given in Table I, and the regression coefficients, with their standard errors in Table II. The values in the last column of Table II are the probabilities (P) that, in the absence of a real effect, values as great as those observed in relation to their standard errors should be obtained by chance. As will be seen, these probabilities are all extremely small, showing that the sample is of adequate size to establish the conclusions. The statistical significance is of course subject to the usual assumptions as to the independence of the cases and their randomness without selection tending to bias the decision. These assumptions are never exactly satisfied, but it is believed that in this case adequate care has been used to make the sample conform to them to a sufficient extent.

A condensed account of our sample is given in Table I, which should be considered if this sample is compared with any other for which the same variates, or any of them, are measured. Our principal results

are embodied in Table II. From this it appears that, among those of a fixed number of preceding pregnancies, and of a given degree of salivary pH, each year of advancing age is accompanied on the average by an increment of approximately 2 in the caries index. But among those of fixed age and pH, those who had had more numerous preg-

TABLE I

Statistics of 324 pregnant women

	MEAN	CORRELATIONS WITH			
		x_1	x_2	x_3	y
x_1 Age	26.06	1.0000	.4959	−.1239	.4728
x_2 Preg. No.	1.95		1.0000	−.4563	.1636
x_3 Salivary pH	6.62			1.0000	−.1531
y Caries	34.53				1.0000

TABLE II

Regression coefficients determined from 324 pregnant women

FACTOR	COEFFICIENT SYMBOL	VALUE OF COEFFICIENT	STANDARD ERROR OF COEFFICIENT	RATIO TO STANDARD ERROR	PROBABILITY P
Age	b_1	2.079	.215	9.66	5.08×10^{-22}
Pregnancy	b_2	−2.819	.966	2.92	.0035
Salivary pH	b_3	−25.06	8.12	3.09	.0020

TABLE III

Regression coefficients from mixed sample of 355, pregnant and non-pregnant

FACTOR	COEFFICIENT SYMBOL	VALUE OF COEFFICIENT	STANDARD ERROR OF COEFFICIENT	PROBABILITY P
Age	b_1	1.781	.182	10^{-21}
Pregnancy	b_2	−1.061	.734	.15
Salivary pH	b_3	35.83	6.26	10^{-10}

nancies were in better dental condition than those who had had few or no previous pregnancies. This effect is so marked that more than 2.8 points in the caries index are on the average to be subtracted for each successive pregnancy. Thus a pregnancy may be said to offset the ravages of more than a year and a third of age, unless some selec-

tive factor intervenes which we have not been able to identify. Certainly there is here no support for the notion that pregnancy destroys the teeth, but rather a marked indication of a beneficial effect. The older idea may perhaps be ascribed to the positive crude correlation between pregnancy number and caries observed in this sample as in others (the value here is about .16, as shown in Table I); but this fails to take account of the important factor of age, which of course has strong positive correlations both with caries and pregnancy number (.47 and .50 respectively). The beneficial effect of pregnancy emerges when, as with our regression equation, suitable account is taken of age and pH.

The negative character of b_3 (the regression coefficient for pH) in Table II points to the tendency that acid mouths are likely to be more carious than those with alkaline reactions. However, this effect is not so important in comparison with age and pregnancy as might be supposed merely by noticing that b_3 is many times greater than b_1 or b_2, (the regression coefficient for age and pregnancy number respectively), since the units of measure are arbitrary. A better idea of the relative effects of these three variables upon caries in the population is obtained by multiplying the values of the coefficients in Table II by the corresponding standard deviations. The products are: for age, 5.47; pregnancy, -3.64; salivary pH, -3.36. These are the regression coefficients which would have been obtained if each of the three variates had been expressed in such units as to have unity as its standard deviation.

Of the total variance of caries in the sample, 25.3 per cent is accounted for by our regression equation. The remaining 74.7 per cent results from differences among individuals arising from causes other than age, pregnancies, and acidity, or from chance, or possibly from nonlinear effects of the three variates considered. The multiple correlation is .50.

The calculation of the regression equation was repeated with the aggregate of the 324 pregnant and the 31 non-pregnant women. The results are given in Table III. In this calculation the value zero was assigned for the value of x_2 (pregnancy number) to each of the 31. It may of course be argued that instead of zero we should have taken a fraction depending upon the fraction of pregnancy elapsed at the time of observation upon the pregnant women, in order to secure compara-

bility. This however is not a serious difficulty, since errors arising in this way are of the same order of magnitude as those resulting from non-linearity, which we neglect.

The mean value of the hydrogen ion concentration x_3 for the 31 non-pregnant cases is 6.720, which is significantly greater than the 6.617 for the pregnant cases. For the primiparae the mean is 6.619, for the multiparae, 6.614, values not significantly different. It is evident that the difference between the pregnant and non-pregnant group has little genuine relation to caries, so far as concerns acidity; the truth seems to be that gastric hyperacidity associated with pregnancy may intensify mouth acidity somewhat. Thus the pregnant group have more acid mouths than the non-pregnant. Also, as explained above, this non-pregnant group had definitely more carious mouths than the pregnant, perhaps on account of selective factors. It must not however be inferred that the pregnant individuals had better teeth because their mouths were more acid. Such a spurious inference would be derived from the positive value obtained for b_3 in Table III if the effect of mixing groups selected in different ways were neglected. The effect of mixing upon the calculated value of b_2 is to reduce it to a non-significant value; but the effect of age indicated in Table III is not greatly different from that in Table II.

The fact that more caries was found in the non-pregnant group with a higher pH of saliva (mean 6.72) than in the pregnant group (saliva pH mean 6.61) indicates that the salivary pH at these levels has little or no influence on caries frequency and may be disregarded as a factor operating during pregnancy. The chief value in this finding is to indicate a tendency; that is if sufficient lowering in salivary pH were present, more caries would result.

As was stated early in the paper, the addition of the non-pregnant group was not essential to the conclusions derived from this study. Table III demonstrates a lack of homogeneity in the two groups and the difficulty encountered when the use of a control group is attempted in a study of this kind. It is chiefly for this purpose that Table III is allowed to remain in this paper. In Table IV the data are summarized.

DISCUSSION

The influence of pregnancy as a possible causative or aggravating factor in dental caries may be considered in relation to the problem

of the causation of caries in general, and to the means by which such influence might be exerted: whether by way of endocrine disturbances accompanying pregnancy and affecting the teeth, or through a disturbance of calcium and phosphorus metabolism.

Recent research indicates that the causes of dental caries include both conditions which operate directly on the environment of the teeth, and other conditions which seem to be metabolic. The environmental factors may include hyperacidity induced by lack of hygiene resulting in bacterial action on food debris thus producing lactic acid, hence more tooth decay. This hypothesis, however, is not supported in the studies of Karshan, Krasnow and Krejci, who found no significant variance in the pH of the saliva of individuals with active caries and caries-free individuals. Another hypothesis concerning hyperacidity is that due to nausea and vomiting during pregnancy, there is an increase in salivary hyperacidity, thus producing more decay. However, from the tables just cited, it is seen that while there was a slight decrease in salivary pH, caries was not increased. Thus, in both the general consideration of caries and its consideration during pregnancy, evidence is lacking as to the etiological rôle of salivary hyperacidity in producing caries.

Turning next to another environmental factor: the studies of Rosebury, Karshan and Foley in rats, and of Rosebury and Karshan among Eskimos, suggest that a primary cause of dental caries in man may be certain fermentable foods which initiate caries directly by virtue of their capacity for forcible impaction into the recesses of the teeth. In rats such foods—e.g., coarsely ground raw cereals—induced caries even though the diet as a whole was fully adequate in all nutritional elements. Diets deficient in calcium and in vitamin D did not produce caries in themselves, but aggrevated caries produced by coarse cereal particles. Dietary deficiency in man seems to have a similar effect, as indicated by the work of numerous investigators. The studies particularly of McBeath, in which ultra-violet irradiation was found to reduce the incidence of caries in children without alteration of the diet, suggest the operation of a metabolic factor. Any effect that may be exerted by pregnancy on caries would probably be part of this latter phenomenon.

From all available evidence it seems clear that pregnancy in itself

could only modify other conditions which cause caries. Waugh and other students of aboriginal races are agreed that Eskimos and other primitive peoples are free from caries in their primitive state, but that decay becomes prevalent among them when they adopt civilized diets. In their primitive state frequent pregnancies do not cause decay, other essential factors being lacking.

In animals it has been shown by Rosebury and Foley that pregnancy and lactation, despite the feeding of diets deficient in calcium and vitamin D, did not cause caries, although calcification of bone and of new dentin was severely defective. It is often assumed that pregnancy may induce removal of calcium from the teeth, as it may from bone; but this is doubtful. In the animal experiments just cited the molar teeth of the mothers, being fully formed when the experiment was started, showed little effect of the deficient diets, and the whole effect was limited to the dentin presumably formed during the experimental period. Albright, Aub and Bauer, reporting their observations of 17 proved cases of hyperparathyroidism, state that "the teeth do not take part in generalized decalcification. They may fall out because of disease of the jaws, but themselves remain well calcified. This is brought out strikingly by roentgenograms in which the well calcified teeth stand out sharply against the poorly calcified jaws. This failure of the teeth to become decalcified is strong evidence against their being a reserve supply of calcium." Our own observations in two cases of this type bear out these observations.

Strock, discussing parathyroid disease, states "at first I thought that the lack of caries was purely incidental, but later, as case after case, both in very young and older ones, showed a lack of caries, it began to seem an important finding." In a personal communication from Strock, he writes, "I have recently seen a patient who has been known to have hyperparathyroidism for three years, who has gone through a pregnancy, in whom there were only two new cavities." Rony, in a discussion of the rôle of the endocrines in producing dental caries supports the idea that hypo- or hyper-function may influence the quality of the teeth during their formative period but after they have attained their final size and structure the enamel and probably the dentin too are not thus affected.

Dental caries linked with such diseases as rickets and scurvy was

considered by Hess and Abramson, in an examination of two groups of children, one group having had rickets in a mild or moderate form in the first year, and other group having been known to be free from rickets. They found that children who had been protected and were free from rickets had developed less caries than those who had not. Nevertheless, numerous instances of caries were found in the non-rachitic group. Deciduous teeth as well as the first molars were involved. However, in other studies by this group, considerable caries was observed in the teeth of a group of children who had been breast-fed and had shown no evidence of rickets, while no caries was observed in the teeth of some of those who had marked signs of rickets, the conclusion being that no parallelism was found between the development of rickets and the subsequent occurrence of caries of the teeth.

Studies concerning themselves with vitamin C deficiencies on the guinea pig show deleterious effects on pregnancy, the bones, and calcification of the teeth. Wells and others observed changes in the odontoblastic layer as well as in the pulp. None, however, reported the development of dental caries.

From the foregoing it is evident that inasmuch as experimental research up to this time records no creditable cause for caries, with the exception of the coarse particle diet cited, there is no basis for designating pregnancy as a cause of dental caries, from the laboratory standpoint. Nor have statistical studies with pregnancy as the basic factor yielded data of a contrary nature.

Gerson examined the teeth of 50 pregnant women and 50 non-pregnant, ages 20 to 30, nulliparous controls, during the second to fourth months of gestation. Six months later she made a re-examination and recorded both superficial and deep caries as well as the number of extractions. Her conclusions are that if pregnant and non-pregnant women have the same number of good teeth to begin with, the harmful influence of pregnancy on the teeth is readily seen at a later period. In an analysis of Gerson's figures so many considerations bearing directly on the problem have been ignored or incompletely touched upon that full credence cannot be given her conclusions. For example, she does not record the number of teeth present to begin with. Hence, her conclusion that "if pregnant and

non-pregnant women have the same number of good teeth to begin with" is merely a postulation. Caries frequency is shown in an age range of ten years—too long a period for comprehensive comparison; the average age, an essential element, is lacking. Progress of decay is measured by the number of extractions necessary in both groups—an erroneous measurement inasmuch as extractions may be necessitated by toothaches or abscesses without progressive tooth decay. Biro reported a study of 200 pregnant women, made up of 103 primiparae, 70 secundiparae, and 27 multiparae. Ages ranged between 16 and 30. In all, 6145 teeth were examined. Caries of more than 9 months standing, less than 9 months standing, total caries and proximal and gingival caries were noted. The results show no significant difference in variation within each group as compared with the variation between the groups. Biro's conclusion is that such differences as there are, are due to disparity in age and not to the number of pregnancies.

Starobinsky reports a study of 216 pregnant and 150 non-pregnant women. He divided the pregnant group (216 cases) into primiparae, (average age 25.8 years) secundiparae (average age 28.6 years), and multiparae (average age 31.4 years). He noted an increase of 4.4 per cent caries of the second over the first and the same increase in the third group over the second. He also divided this same group into three age groups as follows: 20 to 25 (average age 22.5), 25 to 30 (average age 27.1), and 30 to 36 (average age 31.7). The difference in caries percentage shows an increase of 7.5 per cent of the second group over the first, and 5.5 per cent increase of the third group over the second. The 150 non-pregnant, (50 in each age group), he divided into three age groups as follows: 20 to 25 (average age 21.8 years), 25 to 30 (average age 27.3 years) and 30 to 35 (average age 31.2 years). The second age group shows an increase in caries of 7.7 per cent over the first age group, and the third age group over the second of 5.4 per cent. By comparison of the non-pregnant and pregnant groups, it is seen that increase in each age group is about the same, and this increase is larger than was the increase with pregnancy order. It is also significant to note from his study that in the pregnant group, the average age was 27.5 years with an average caries of 26.7, while in the non-pregnant group the average age was 26.7 years with an aver-

age caries of 28 per cent. Starobinsky concludes that since the average yearly increase in carious teeth in the pregnant group was about 1½ per cent and the average increase in the non-pregnant group was about 1½ per cent, pregnancy must be ruled out as a cause of the increase.

In a study of Ziskin, similar findings were noted. The pregnant group (599 cases) showed an average of carious teeth of 35 per cent (average age 25.83 years) and the never pregnant group (205 cases) showed average carious teeth 43 per cent (average age 27.05 years).

Studying the problem from still another angle, we took case histories on 154 of the 324 pregnant women with whom the present report concerns itself. Of these, (1) there were 46 in whom there were no

TABLE IV

Averages for entire group

	AGE	CARIES INDEX	CARIES INDEX PER YEAR	CARIES INDEX PER YEAR AFTER 6 YEARS OF AGE	PER CENT CARIOUS SURFACES PER YEAR AFTER DEDUCTING VALUES FOR FILLINGS AND MISSING TEETH
O Pregnancy	27.7	42.9	1.54	1.97	7.9%
Primiparae	23.6	31.0	1.31	1.75	5.3%
Secundiparae	27.1	35.9	1.32	1.70	5.1%
Multiparae	29.1	39.0	1.31	1.64	6.7%

new cavities present; (2) 31 of the women were aware of their cavities before pregnancy, in some the cavities having been present from two to four years; (3) 30 had new cavities—mainly pit and fissure cavities that the patient could not feel with her tongue or by the retention of food debris. However, although it is likely that some cavities in the latter group were present before the pregnancy, owing to the indefinite histories they are classed as new decay; (4) 47 women were not certain of all their cavities. Some, they knew, were present before pregnancy, others they were unaware of. These are placed in the questionable group. The average length of time that elapsed between the last visit to a dentist and our examination was from two to two and a half years.

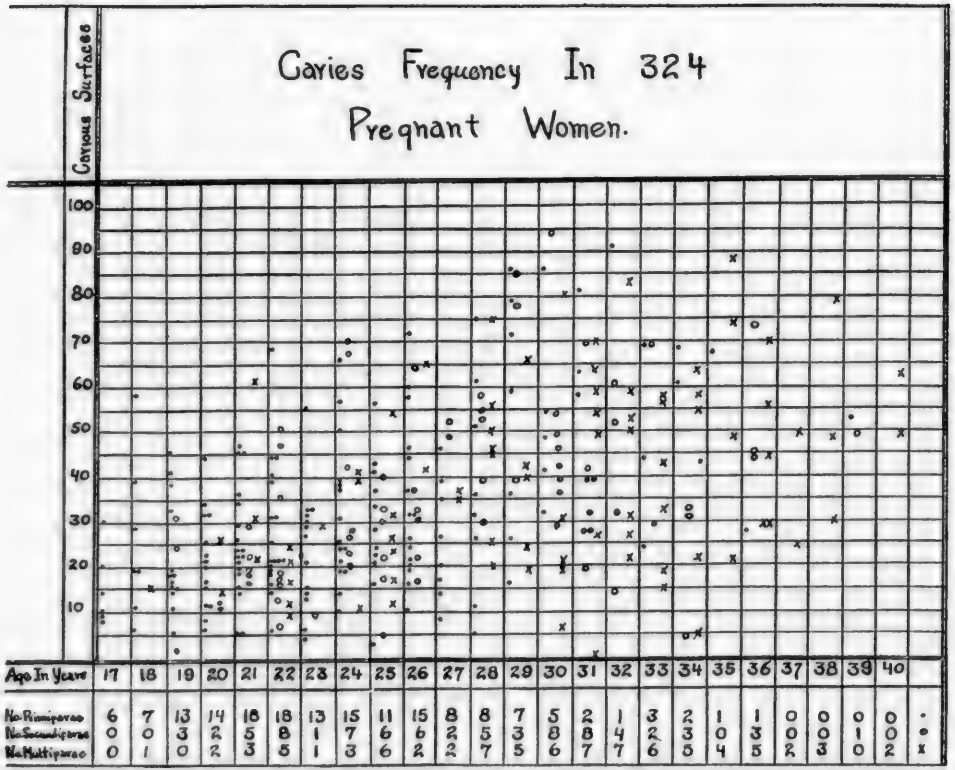
Caries Frequency In 324
Pregnant Women.
Carious Surfaces
100
90
80
70
60
50
40
30
20
10
Age In Years 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40
No Primiparae 6 7 13 14 16 18 13 15 11 15 8 8 7 5 2 1 3 2 1 1 0 0 0 0 .
No Secundiparae 0 0 3 2 5 8 1 7 6 6 2 5 3 8 8 4 2 3 0 3 0 0 1 0 •
No Multiparae 0 1 0 2 3 5 1 3 6 2 2 7 5 6 7 7 6 5 4 5 2 3 0 2 x

FIG. 2

It has been our observation that teeth with large cavities may be free from pain for some time before the pregnant state, but become painful during the term, necessitating extraction. Starobinsky sees an explanation of this in that "the hyperemia of the head usually present in pregnancy evokes a hyperemia of the dental pulp, and on this account the toothache, whether due to caries or chronic pulpitis, is the more violent." Bumm (quoted by Starobinsky) seeks the cause in the congestion of abnormal products of metabolism, and Tanzer (quoted by same author) in the increased intradental blood pressure.

Another question which arises is whether or not the apparent decrease in caries frequency may not be explained by the fact that these women, coming to the clinic, were receiving dietary instruction. Since our examinations were conducted during routine prenatal care, usually when patient first entered the clinic, this factor cannot be considered as having any bearing. It may be further pointed out that Biro's observations which support those cited here, were published in the year 1899.

SUMMARY AND CONCLUSIONS

This study is based on a group of 324 pregnant women, with pregnancies ranging from one to eight months. By the method of least squares, statistical analysis of the data shows that pregnancy not only does *not incite* tooth decay but that some factors operating during pregnancy actually prevent tooth decay to a significant extent. Literature is cited dealing with the problem of dental caries generally from the standpoint of local environmental etiological factors and metabolic. The environmental factors include a consideration of hyperacidity of the saliva and diets consisting of coarse particles. The metabolic studies include a consideration of dental caries in such diseases as rickets and scurvy and imbalance of the endocrines. The status of the teeth of Eskimos and other aboriginal peoples are dealt with. Other statistical studies on the occurrence of dental caries in pregnant women are listed and discussed. The observation is made that more teeth may be extracted during pregnancy than before the gravid state, but such extractions may become necessary for reasons other than progress of tooth decay.

From the foregoing we conclude: (1) That pregnancy, *per se*, is

not a cause of dental caries, (2) That while the saliva is slightly more acid during pregnancy, the degree of acidity is not sufficient to produce tooth decay, (3) That some factors operating during pregnancy actually prevent tooth decay to a significant extent.

We wish to express our thanks to Dr. Theodor Rosebury, College of Physicians and Surgeons, Columbia University.

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BACTEREMIAS FOLLOWING DENTAL EXTRACTION. DEMONSTRATION OF SOURCE OF BACTERIA BY MEANS OF A NON-PATHOGEN (SERRATIA MARCESENS)[1]

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This study was undertaken to determine the frequency of bacteremias following the extraction of teeth and to demonstrate the original location of the bacteria which were recovered from the blood stream. Other objectives were to determine how different anesthesias and how the preparation of the gingival crevice with one-half strength tincture of iodine effected these results.

In studies dealing with the etiology of subacute bacterial endocarditis, Okell and Elliot (1) made blood cultures before and ten minutes after the removal of teeth under general anesthesia. They obtained over 60 per cent positive blood cultures after extraction under nitrous oxide. No serious systemic manifestations are recorded in spite of the high incidence of transient bacteremias. In several cases of severe pyorrheal disease, positive blood cultures were obtained prior to extraction.

More recently, Round, Kirkpatrick and Hails (2) made studies on 90 cases in which novocaine infiltration anesthesia was used. The technical methods were devised to prevent possible contamination of the needle or the anesthetic solution and care was taken to prepare the site of injection although no mention was made of similar preparation of the gingival crevice. The results of post-operative blood cultures were not stated. These investigators took blood cultures on ten individuals with oral sepsis who had just masticated hard candy. The two cases of pyorrheal disease showed a bacteremia, *Staphylococcus aureus* being obtained from one and *Streptococcus*

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viridans from the other. Earlier studies made by Round and Kirkpatrick (3) and Broderick and Round (4) were concerned with the presence of bacteria in the submucous tissues. They thought many of the sequellae following novocaine anesthesia were due to the spread of organisms from the sub-mucous tissues when the novocaine was injected. Although it is not specifically stated in these papers, it is inferred that these organisms in the submucous tissues may be of significance in producing bacteremias.

The previous studies in this field then have been concerned with: (a) The presence of organisms normally in the sub-mucous tissues, (b) The frequency of bacteremias following extraction of teeth under general anesthesia, and (c) The frequency of bacteremias following mastication. From Kirkpatrick's papers one gathers the impression that organisms in the submucous tissues may be partly responsible for the bacteremias. On the other hand, Okell and Elliot's work indicates a rough relation between the amount of oral infection and the percentage of positive blood cultures. Apparently no attempts have been made to sterilize the gingival crevice prior to the removal of the tooth. Periapical cultures have not been made of the extracted teeth for comparison with the organisms present in the blood stream, nor has the anatomical site of the organisms, later demonstrated in the blood stream, been determined. Serological studies with the organisms recovered from the blood stream and the patient's serum have not been reported.

The subjects for this study were dental admittances of the outpatient clinic of the New Haven Hospital. Control cultures were taken from attending staff members and laboratory assistants. The blood for bacteriological examination was secured by venepuncture in the antecubital region. Sterile gauze sponges moistened with tincture of iodine were swabbed over this region followed by 70 per cent ethyl alcohol similarly applied. Twenty cc. glass syringes were used routinely with $1\frac{1}{2}$ inch, 21 gauge steel needles or $\frac{1}{2}$ inch, 26 gauge needles if difficulty was experienced in entering the vein. Ten cc. of blood were withdrawn for culturing; an additional 2 cc. were withdrawn for serological studies in selected cases. Two cc. of the blood were ejected into cooked ground meat media and the remainder into beef infusion broth.

When novocaine infiltration or conduction anesthesia was employed the mucous membrane at the site of injection was sterilized with 3.5 per cent alcoholic iodine solution after which about 2 cc. of 2 per cent novocaine with 1:20,000 adrenalin were injected. In addition to the clinical condition of the tooth, general gingival conditions, type of injection, duration and severity of the operative treatment and the time elapsing before the blood was cultured, were recorded. An attempt was made to perform the venepuncture 10 minutes after removal of the tooth.

The patients were placed in four general groups on the basis of the procedures which had been employed prior to the venepuncture.

Group I comprised 53 observations which included bacteriological studies of the gingiva, tooth apex, socket and blood stream. Prior to extraction a culture was made of the gingiva. A sterile No. 18 dental excavator was used to obtain the material which was inoculated on a surface agar plate containing 5 per cent rabbit's blood. One-half strength tincture of iodine was then applied to the gingival crevice by means of small cotton swabs and after an interval of 30 seconds to a minute, a gingival culture was again taken in the same manner, and inoculated on a second blood agar plate. An agar slant suspension of *Serratia marcesens* (*B. prodigiosus*) in 10 cc. of normal saline was then painted around the neck of the tooth to be removed, with a small cotton applicator and repeat cultures taken of the gingival crevice by the same procedure and using the same media. The tooth was extracted with sterile forceps with a minimal amount of trauma and the tooth apex gently streaked over a fourth blood agar plate. Immediately after the removal of the tooth, a small sterile swab was inserted in the alveolus to absorb the first drop of blood welling into the socket. This was streaked over a fifth blood agar plate.

Group II consisted of 92 observations in which the gingiva received no preparation prior to the extraction. Aside from the clinical data recorded for each patient no experimental procedures were employed other than the venepuncture.

Group III comprised 37 observations in which the gingiva was painted with a saline suspension of *Serratia marcesens* just prior to the removal of the tooth. Blood cultures were taken approximately ten minutes later.

Group IV comprised 21 observations made on clinic patients prior to operative treatment or on attending staff members who had no known systemic disease.

In all instances where positive blood cultures were obtained an attempt was made to secure another blood culture at a later date.

DATA

Group I, which was studied in greatest detail, comprises data obtained from 53 blood cultures, 56 periapical and 55 gingival cultures. Positive blood cultures were obtained in 12 cases (22.6 per cent) of the examinations. Positive blood cultures were obtained from 46 per cent (15) of the conduction injections and 13 per cent (38) of the

infiltration injections. Conduction injections were limited to mandibular block. The frequency of the different bacteria isolated from the blood stream in Group I is given in Table I.

The following mixed cultures were obtained in this group: (a) *Pneumococcus, Streptococcus viridans*, diphtheroid, (b) *Staphylococcus aureus* and *Streptococcus viridans* (R & S).

Gingival cultures made from 55 teeth showed the usual mouth flora in every instance. After the application of one half strength tincture of iodine, mixed cultures of the common mouth organisms were obtained from 18 of the gingivae, 21 were positive for streptococci, usually of the viridans variety, and 16 were negative. The majority of the negative results were obtained from mandibular teeth.

TABLE I

Frequency of bacteria isolated from blood stream in group I cases

Bacteria	No. Times	Positive Bacteremias (12) (per cent)
Diphtheroids	5	41.5
Serratia marcesens	4	33.3
Strep. non-hemolyticus	2	16.6
Staph. aureus	2	16.6
Pneumococcus	1	8.3
Gram-negative non-spore forming rod	1	8.3

The preparation of the gingiva with iodine and the gingival smears were made by several different operators. Gingival cultures taken after the application of *Serratia marcesens* were positive for this bacterium in all but 8 instances. In all but 3 of these cases no growth was obtained from the gingival smears taken after the application of iodine.

The apexes of 56 extracted teeth were examined bacteriologically and positive cultures for *Streptococcus viridans* were obtained in 43 instances, *Serratia marcesens* was recovered from 30 apexes and other organisms from 9 apexes. *Staphylococcus aureus* was cultured from 3 apexes when the gingival smears following the application of iodine were negative for this organism. Since *Serratia marcesens* was painted about the gingival margin immediately prior to extraction it was interesting to note that 25 of the 56 apical cultures were negative

for this organism; 18 of this group, however, were positive for *Streptococcus viridans*.

Group II, comprising 92 observations, includes cases where novocaine or nitrous oxide-oxygen anesthesia, without other experimental procedures, were used for the removal of the teeth. Conduction anesthesia was employed for 40 extractions, novocaine infiltration for 46, nitrous oxide for 1; 5 cases received no form of anesthesia. The frequency of positive cultures when the various types of anesthesia were employed is given in Table II.

TABLE II

Frequency of positive cultures in group II with various types of anesthesia

Type	No. Observations	No. Positive	Positive Cultures (per cent)
Conduction	40	6	15
Infiltration	46	3	6.5
No anesthesia	5	1*	20
Nitrous oxide	1	1*	100

* Acute alveolar abscess.

TABLE III

Frequency of organisms isolated from blood stream in group II cases

Organism	No. Times Recovered	Positive Bacteremias (11) (per cent)
Staphylococcus aureus	7	63.6
Streptococcus viridans	4	36.3
Streptococcus non-hemolyticus	1	9
Gram-negative rod	1	9

Although conduction and infiltration anesthesia were used with nearly equal frequency, twice as many positive cultures were obtained after conduction.

The frequency of the organisms recovered from the blood stream in Group II cases is given in Table III.

The following mixed cultures were obtained in this group: (a) *Staphylococcus aureus*, Gram-negative rod. (b) *Staphylococcus aureus*, *Streptococcus viridans*.

In *Group III*, 37 cases were studied, conduction anesthesia being used for 19 extractions and novocaine infiltration for the remainder. The gingival crevice was painted with a heavy suspension of *Serratia*

marcesens in normal saline just prior to extraction. Positive findings were obtained 5 times when conduction anesthesia was employed and 9 times when infiltration was used. Other bacteria were found 3 times following conduction anesthesia and 4 times following infiltration. The frequency of the organisms obtained from the blood stream in Group III cases is given in Table IV.

The following mixed cultures were obtained in this group: (a) 2 *Serratia marcesens, Streptococcus non-hemolyticus,* (b) *Serratia marcesens, Streptococcus non-hemolyticus* and diphtheroid.

TABLE IV

Frequency of organisms isolated from the blood stream in group III cases

Organism	No. Times Recovered	Positive Bacteremias (17) (per cent)
Serratia marcesens	14	82.4
Streptococcus non-hemolyticus	5	29.4
Diphtheroids	1	5.9
Gram-negative coccus	1	5.9

TABLE V

Summary of positive blood cultures compared with the type of anesthesia used

Group	No. Observ.	No. Cond. Anesthesia	Per cent Positive	No. Infil. Anesthesia	Per cent Positive	Total Positive Cultures (per cent)
I	53	(15)	46	(38)	13	22.6
II	92	(40)	15	(46)	6.5	11.9
III	37	(19)	26	(18)	50	37.8
IV	21	—		—		4.7

One positive result was obtained from the 21 examinations in *Group IV*, which comprised the control series. The bacteremia (Streptococcus non-hemolyticus) was demonstrated in an individual with a moderate degree of gingival infection but who had no known foci of infection. Interesting results were obtained in three other cases which were not included in the control group. The gingival tissues of a patient with a moderate degree of generalized pyorrhea were massaged digitally for 5 minutes. *Staphylococcus aureus* and *Streptococcus viridans* were cultured from blood withdrawn ten minutes later. A non-hemolytic streptococcus was grown from the blood stream in a case of osteomyelitis of the mandible with accompanying cellulitis of the soft tissues.

A general summary of the frequency of demonstrable bacteremias compared with the type of injection used is given in Table V.

The test organism, *Serratia marcesens*, was painted about the gingiva prior to extraction in groups I and III. Bacteremias of organisms other than *Serratia marcesens* were demonstrated in 16.9 per cent of the examinations made on cases where teeth had been removed.

The type of injection used and the amount of operative trauma produced during extraction were compared for the cases showing bacteremias with *Serratia marcesens* and for those in which other organisms were demonstrated in the blood stream. All simple ex-

TABLE VI

Comparison of type of injection employed and degree of operative trauma with incidence of bacteremias (per cent)

Blood Stream	Conduction	Anesthesia Infiltration	Operative Trauma +	++	+++	++++
Negative	77.0	77.5	75	9	11	5
Serratia marcesens	9.4	10.8	47	47	6	—
Other organisms	13.5	12.0	57	35	8	—

TABLE VII

Comparison of the degree of gingival infection and the clinical condition of the tooth with the incidence of bacteremias (per cent)

Blood Stream	Gingiva Poor	Fair	Good	Caries	Crown Pyorrhea	Roots
Negative	20	74	6	61	19	19
Serratia marcesens	42	52	6	65	15	20
Other organisms	24	64	16	65	24	10

tractions were classed as one plus. Difficult extractions or fractured roots were classified according to the amount of elevating and operative trauma inflicted as well as the duration of the operative procedure. These findings are summarized in Table VI.

There were no marked differences observed in the type of anesthesia or the degree of operative trauma when bacteremias of *Serratia marcesens* or other organisms were demonstrated. These findings may suggest that similar factors and avenues of invasion into the blood stream play a rôle in the production of all the bacteremias.

The incidence of bacteremias was compared with the degree of gingival infection and the clinical condition of the crowns of the teeth. These results are given in Table VII.

Additional studies will be required to determine the significance of trends indicated by these findings. It it probable that both the degree of oral sepsis and the amount of operative trauma are of significance in the production of bacteremias.

Repeat blood cultures were obtained in 20 cases. Five negative repeat examinations were obtained from as many negative original cultures. All the remaining repeat cultures were made on patients who had had a bacteremia previously. Two of the second cultures were positive but a third culture was negative in both cases. The duration of the bacteremias was not determined. Repeated cultures in the case of *Serratia marcesens* bacteremias were always sterile and no serological changes specific for this organism were observed in the blood serum of these individuals. No ill effects were described by any of the individuals with *Serratia marcesens* bacteremias.

DISCUSSION

Omitting the bacteremias of *Serratia marcesens*, positive blood cultures were demonstrated in 17 per cent of the 204 cases when novocaine anesthesia was used. This incidence of bacteremias is much lower than that reported by Okell and Elliot (60-76 per cent); however, these investigators employed nitrous oxide for all extractions and do not indicate that they treated the gingiva in any special way.

The use of general anesthesia or differences in the physical condition of the patient may be important factors in the production of transient bacteremias. Similar procedures carried out on individuals such as those studied by Okell and Elliot and subjects from the outpatient clinic would probably give different findings but it is doubtful if this would account for the differences in the frequency of bacteremias reported by Okell and Elliot and that obtained in the present investigation.

The novocaine anesthesia may be partly responsible for the lower incidence of positive blood cultures. The adrenalin in the anesthetic solution produces a marked capillary constriction, sufficient perhaps to serve as an effective barrier against the mechanical forcing of bacteria into the blood tream. This ischemic effect would be most marked in infiltration injections and less marked in the conduction or "block" type of injection. Additional studies will be required to

determine the significance of the vaso-constricting action of adrenalin in reducing the frequency of bacteremias. The *Serratia marcesens* bacteremias resulted in spite of the capillary constriction produced by the adrenalin.

The mechanism by which all of the organisms enter the blood stream is not yet established definitely. The investigations of Round and Broderick suggested that, in the case of injection anesthesia, the original site of some of the organisms may be in the submucous tissues. Okell and Elliot found that the incidence of bacteremias was related to the degree of oral sepsis. The use of *Serratia marcesens* in the present study demonstrated that organisms in the gingival crevice can be forced into the vascular system during extraction. Gingival massage in individuals with pyorrheal disease was sufficient to produce a transient bacteremia.

Since the *Serratia marcesens* bacteremias definitely were not due to organisms being forced into the vascular channels during the novocaine injection they must have resulted from organisms in the gingival crevice being forced into the blood stream by the piston-like action of the tooth during extraction. This finding emphasizes the necessity for more effective methods of sterilizing the gingival crevice prior to extraction.

A tabulation of the clinical gingival findings and the type of anesthesia used (infiltration or conduction) with the bacteriological results revealed no striking differences in the clinical findings when *Serratia marcesens* bacteremias or those with other organisms ensued. It is possible that insufficient variation in the degree of oral sepsis was present in the cases studied to demonstrate the association between oral sepsis and incidence of bacteremias which Okell and Elliot observed.

In groups I and II a higher percentage of bacteremias was demonstrated in conduction injected cases. Whether this finding was dependent on the type of injection *per se* or the fact that it was used only in the lower jaw is not known. Analysis of deaths following dental infections or infections following injection anesthesia reveal that the opportunity for spread of infection is greater in the lower jaw, especially in the region of the pterygoid fossa. Many of the infiltration injected bacteremias in group III were positive for *Serratia*

marcesens. This result was independent of the type of injection used and was due entirely to operative trauma.

SUMMARY

Bacteremias of organisms other than *Serratia marcesens* were demonstrated in 16.9 per cent of the cases after the removal of teeth under novocaine anesthesia. The frequency of bacteremias could not be associated definitely with the degree of oral sepsis or the severity of the operative procedure. The inability to correlate the incidence of bacteremias with the degree of oral sepsis may be explained by the majority of the cases falling in one clinical division.

Repeated blood cultures revealed that the bacteremias were transient. No specific serological changes in the blood stream were observed following bacteremias with *Serratia marcesens* or other organisms.

The lower incidence of bacteremias (17 per cent), as compared with Okell and Elliot's findings (60–76 per cent) may be due to the use of injection anesthesia in which 1:20,000 adrenalin plays a significant rôle as a vaso-constricting agent.

Attempts to sterilize the gingival crevice revealed the inadequacy of the method and antiseptic used. Although the bacterial flora was quantitatively markedly reduced after treatment, in many instances pure cultures of *Streptococcus viridans* were grown from the gingival crevice. This may be of significance in the interpretation of periapical cultures made on extracted teeth.

The use of *Serratia marcesens* demonstrated that organisms in the gingival crevice can be forced into the blood stream during dental extractions. This observation emphasizes the necessity for improved methods of sterilizing the gingival crevice prior to extraction, and caution in performing intra-oral operative procedures on chronically ill individuals.

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EFFECT OF CERTAIN MOUTHWASHES ON THE NUMBER OF ORAL BACTERIA[1]

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The purpose of this study was to determine the duration of the inhibitory effect of some of the popular mouthwashes on the oral bacteria. All of the preparations used with the exception of one, Azochloramid, were bought in the open market. Azochloramid is a new preparation and was supplied to us by the manufacturer.

Two experiments were carried out. In the first, Pepsodent, Lavoris, and Hexylresorcinol were diluted 1:1 with water, while Azochloramid was made up by dissolving one tablet in 50 cc. of water. This dilution of the first four products named was used because all of the manufacturers of these products advertise that dilutions of that strength can be used, as well as weaker dilutions. Since these were preliminary tests, we used the greater concentration which should be the most efficacious. Tests were made on six individuals, but not all mouthwashes were used on all individuals.

In the second experiment, Azochloramid—1 tablet in 50 cc. of water, Chlorozene—1 tablet dissolved in 125 cc. water, Zonite—1:10 dilution and Pepsodent—1:3 dilution were used. Tests were made on two individuals, one of whom (M. C.) had been tested in Experiment I.

The methods of sampling were identical in both experiments and were the same as reported in a previous publication.[2] Briefly, samples were collected by brushing the teeth and then rinsing both mouth and toothbrush with N/300 NaOH. This material was sprayed through an atomizer especially devised to break up the larger clumps. The volume of the sprayed material was made up to 20 cc. with N/300 NaOH and 0.01 cc. portions smeared out on a glass slide, stained by

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[2] Crowley, M. C. and Rickert, U. G., *J. Bact.* 30: 4, 1935.

Gram's method, and counted. In this study, N/300 NaOH was substituted for N/400 NaOH as reported in the previous publication. Food was restricted to fluids during an eight hour period on test days. The initial sample was collected two hours after breakfast. Immediately after the first sample was collected, 15 cc. of the preparation to be tested was swished thoroughly in the mouth for 1 minute. Counts were then made at 3 and 6 hour intervals after the use of the mouthwash. The same procedure was carried out on days in which control counts were made with the exception that no mouthwash was used. Control counts were made in order to determine the normal count fluctuations in the individual. Counts for controls and mouthwashes were made at the same time of day on different days. At least 1 day intervened between counts. In these cases neither caries nor gingivitis were clinically demonstrable. In order that the normal oscillation of counts would not confuse their interpretation, only averages of at least 3 were considered, except Listerine and Hexylresorcinol in M. K. (See Table I.) This means that at least 72 counts were made for each mouthwash tested and for each control.

In the past it has been customary to compare the counts obtained after the use of the materials tested with the initial count which was made before the use of the mouthwash. The initial counts, however, were found to be unsatisfactory controls because of their normal fluctuation. We, therefore, in this study compared the average of counts taken on treatment days, with the average of counts made on control days. The accompanying tables are constructed on the basis of percentage variation from the initial count on treatment and control days.

These tables show the number of counts made on each mouthwash, averages of the counts made before the use of the mouthwash, three hours and six hours after the use of the mouthwash, with the decrease or increase from the initial account expressed in per cent.

Experiment I—Table I. In the case of M. C., Pepsodent in a 1:1 dilution showed the greatest decrease, a difference of about 40 per cent from the control count. Azochloramid showed a good reduction. In the case of M. K., Azochloramid and Pepsodent were good. With F. H., Lavoris showed a big decrease at 3 hours but not much at 6 hrs.

Pepsodent 1:1 was good. Listerine showed an increase after being used. The remainder of the table shows cases in which only a few

TABLE I

Experiment I, Effect of mouth washes as compared to control counts

NAME	MOUTH WASH USED	NUMBER OF COUNTS	AVERAGE OF TOTAL NO. OF BACTERIA REMOVED FROM MOUTH*			VARIATION FROM INITIAL COUNT	
			Initial count	3 hrs. later	6 hrs. later	3 hrs. later	6 hrs. later
						(*per cent*)	(*per cent*)
M. C.	Azochloramid	5	5,110	2,620	2,840	−49	−45
	Pepsodent	7	4,120	1,690	1,800	−59	−57
	Lavoris	5	4,630	2,560	3,260	−45	−30
	Listerine	5	5,550	3,990	3,440	−28	−39
	Hexylresorcinol	4	4,230	2,780	2,780	−35	−35
	Control	10	4,360	3,770	3,740	−14	−15
M. K.	Azochloramid	3	5,610	2,560	3,070	−55	−46
	Pepsodent	3	3,630	2,490	2,440	−32	−30
	Lavoris	3	2,980	2,080	2,670	−31	−11
	Listerine	2	2,760	2,100	3,120	−24	+12
	Hexylresorcinol	2	3,060	2,620	3,000	−15	−2
	Control	7	3,890	3,090	3,480	−21	−11
F. H.	Azochloramid	5	3,780	2,320	2,560	−39	−33
	Pepsodent	6	4,660	2,500	2,790	−47	−41
	Lavoris	5	6,280	2,720	5,620	−57	−11
	Listerine	5	3,980	3,990	4,610	0	+15
	Hexylresorcinol	5	5,830	3,340	4,840	−43	−17
	Control	10	4,340	4,110	5,420	−6	+24
F.	Hexylresorcinol	3	1,790	1,123	1,340	−38	−26
	Control	4	2,350	2,400	2,400	+2	+2
O. Z.	Pepsodent	3	8,900	7,290	4,800	−19	−48
	Listerine	3	4,440	4,940	4,370	+11	−2
	Control	3	7,320	9,030	8,890	+23	+21
C. K.	Azochloramid	3	6,530	3,910	3,670	−41	−44
	Control	3	6,220	4,930	4,690	−21	−35

* Expressed in millions.

antiseptics were tested on each individual. In the case of O. Z., Pepsodent 1:1 showed a decrease. The other cases do not show

enough of a decrease to have any significance. It is interesting to see also that Listerine with O. Z., as in the case with F. H., shows an increase.

Experiment II—Table II. In the case of M. L. not one mouthwash showed a *marked* decrease. With M. C. the same is true. The most interesting thing about M. C. is the difference in counts between the controls of the two experiments. In the second experiment we have a decrease of 30 per cent at the end of 3 hours and 44 per cent at the end of 6 hours while in the first experiment, as pointed out in Table I,

TABLE II

Experiment II, Effect of mouth washes as compared to control counts

NAME	MOUTH WASH USED	NUMBER OF COUNTS	AVERAGE OF TOTAL NO. OF BACTERIA REMOVED FROM MOUTH*			VARIATION FROM INITIAL COUNT	
			Initial count	3 hrs. later	6 hrs. later	3 hrs. later	6 hrs. later
						(*per cent*)	(*per cent*)
M. L.	Azochloramid	3	4,080	3,070	3,020	−25	−26
	Zonite	3	4,150	2,580	2,620	−38	−37
	Chlorazene	3	5,070	2,810	2,580	−45	−49
	Pepsodent 1–3	3	5,310	2,830	3,040	−47	−43
	Control	5	4,840	3,770	3,840	−23	−21
M. C.	Azochloramid	3	6,130	3,050	3,320	−51	−46
	Zonite	3	6,450	3,120	3,510	−48	−46
	Chlorazene	3	6,210	3,680	3,140	−41	−50
	Pepsodent 1–3	3	6,890	4,230	4,790	−39	−28
	Control	5	7,030	4,940	3,950	−30	−44

* Expressed as millions.

the decrease was only 14 per cent at 3 hours and 15 per cent at 6 hours. This is a good example of the difficulty in judging tests of this type.

DISCUSSION

The results of these experiments show much individual variation and reaction to the separate mouthwashes. The type of organism predominating in the mouth may play a part. The organisms in the mouth of M. K. were predominately gram negative and here Azochloramid gave the best results. In M. C., Experiment I, where gram positive cocci predominated, both Pepsodent 1:1 and Azo-

chloramid reduced the number of organisms materially but in Experiment II, where the oral flora had changed to one in which gram positive rods (*B. acidophilus*) predominated, no one of the antiseptics used decreased materially the control count which was much higher than in Experiment I.

While some of these antiseptics gave good results as far as reducing the oral flora was concerned, there are other reasons which would mitigate against their use. Pepsodent in a 1:1 dilution and Hexylresorcinol at the same dilution when held in the mouth for one minute caused a burning sensation. Hexylresorcinol at this concentration was only a mild antiseptic. Pepsodent at a 1:3 dilution did not give extremely satisfying results. Chlorozene and Zonite besides not giving marked reductions in the only two cases tested, have too unpleasant a taste to have much of a popular appeal. Azochloramid while not having an obnoxious taste was only fairly antiseptic.

In our opinion no definite conclusions can be drawn as to the antiseptic value of the mouthwashes tested because of the great variation in the same individual from time to time in both control and experimental tests. Count methods are not to be relied upon in testing mouthwashes.

It might be stated here that in the light of our present limited knowledge in oral bacteriology, we cannot assume that a mouthwash effective in reducing oral flora is advantageous in maintaining good oral health. We do not know but that by depressing certain harmless bacteria we may be actually lowering the resistance of the individual against disease producing forms.

TITLE ABBREVIATIONS FOR AMERICAN DENTAL PERIODICALS AND BIBLIOGRAPHIC METHODS[1]

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The inadequacies of dental journalism in that phase of bibliographic method which relates to the abbreviation of the titles of periodicals frequently has been noted and commented upon. Little has been done by the dental profession, *collectively*, to improve this condition. As a result, much confusion and inaccuracy prevails.

Certain constructive organizations in the profession are beginning to recognize their responsibilities in this matter. Gies (1), speaking before the American Association of Dental Editors at San Francisco in 1936, brought this matter to the attention of a representative body of dental editors. He stated: "Uniformity in the journalistic use of abbreviations for the names of periodicals, and for related bibliographic data, is very desirable. Uniformity would promote accuracy, assure definiteness, and serve convenience. Agreement as to the most acceptable uniformity should be achieved and observed."

Uniformity in this matter is of importance to research workers, editors, and librarians. To scientific investigators: because they should be assured of accuracy in their references, ease in interpreting them, and convenience in locating them. To editors: because they have a responsibility to their readers in making available and useful the bibliographic material they present. To librarians: because uniformity is essential in a card index, to say nothing of other desirabilities encountered in routine library procedures.

SOURCES AND METHODS

In preparing this study these procedures were followed: (*a*) reference was made to the best, established, American sources for periodical

[1] Presented at the 15th Annual Meeting of the International Association for Dental Research, Baltimore, March 13–14, 1937.

[2] Chairman of the Committee on Abbreviations and Vice-President of the American Association of Dental Editors.

abbreviations and bibliographic data; (*b*) these source-listings were studied, and their adaptability to dental periodicals noted; (*c*) a list of basic abbreviations was compiled, more or less consistent with these sources where warranted; (*d*) this list was then applied to existing American dental periodicals for illustrative purposes, and (*e*) similar methods were followed in relation to bibliographic data having to do with references and citations. Instructive information, leading data, and in some instances listings were obtained from the following organizations, publishers, and individuals, either through direct correspondence or by reference to published works: Library of the American Medical Association (2), United States Army Medical Library (3), University of Chicago Press (4), B. W. Weinberger (5), American Library Association, Medical Library Association, H. W. Wilson Company, New York: publishers of various indexes and reference works (6), R. R. Bowker Company, New York: publishers of *The Library Journal*, and several standard dictionaries and thesauri.

In studying these source-listings and recommended data, it was found that in addition to some minor consistencies, they were more or less agreed that abbreviations be prepared as far as possible in accordance with the following principles: (*a*) to follow the exact order of the words of the title; (*b*) to make them as brief as is consistent with clearness to those who are familiar with the special literature involved, and (*c*) to attain uniformity. Concerning this last rule—the attainment of uniformity—these sources either state or imply that some exceptions are found expedient. The *Index-Catalogue* says, "The convenience of the reader is regarded as of more importance than a rigid adherence to uniformity." That principle must be remembered in compiling and discussing any listing of abbreviations for the titles of periodicals.

Also to be kept in mind is the fact that nearly all abbreviations in *combinations*, are clear, even when very brief, and that such a *combination* will almost automatically suggest the *full title*. It is conceivable and admitted that some of the abbreviations used in the basic listing presented here, if *isolated*, might not be clear. But in analysing any particular feature of this listing, it must be distinctly understood that only abbreviations in combinations are being considered.

BASIC ABBREVIATIONS

This listing was compiled after a study of the sources mentioned, as well as the abbreviations now in more or less common usage. It contains those words which occur in the titles of dental periodicals (American) now being published, together with some few being used in allied scientific fields.

Abstract	Abstr.
Academy	Acad.
Alumni	Alumni
American	Am.
Annals	Ann.
Archives	Arch.
Assistants	Asst.
Association	A.
Bulletin	Bul.
Bureau	Bur.
Canadian	Canad.
Clinical	Clin.
College	Col.
Congress	Cong.
Cosmos	Cosmos
County	Co.
Dental	D.
Dentistry	Den.
Department	Dept.
Digest	Digest
District	Dist.
Eastern	East.
Education	Educ.
Experimental	Exper.
Gazette	Gaz.
Graduate	Grad.
Health	Health
Hygiene	Hyg.
Institute	Inst.
International	Internat.
Journal	J.
Laboratory	Lab.
Magazine	Mag.
Medical	M.
Medicine	Med.
Modern	Mod.
National	Nat.
Northern	North.
Odontological	Odont.
Orthodontia	Orthodont.
Photography	Photog.
Proceedings	Proc.
Public	Pub.
Quarterly	Quar.
Radiography	Radiog.
Radiology	Radiol.
Record	Rec.
Report	Rep.
Research	Res.
Review	Rev.
Science	Sc.
Scientific	Scient.
Section	Sect.
Society	Soc.
Southern	South.
Supplement	Suppl.
Surgery	Surg.
Transactions	Tr.
University	Univ.
Western	West.

Some of the abbreviations contained in the preceding list differ from those now in use, both in dental and the allied medical and scientific works. There has been an attempt to improve any abbreviation even where generally accepted; the attitude in this respect, which was deemed proper, is expressed in this question, "Was the basis for the original selection poor?" That question was asked about every abbreviation. As a result, the listing deviates in some instances from present usage, and the reasons for the proposed changes are explained.

1. *American.* This is commonly abbreviated *Am.*, and as such is sufficiently understandable. However, one exception is suggested: when the reference is directly to the publications of the American Dental Association, then it be abbreviated simply as *A.* Hence, the *Journal of the American Dental Association* will be *J. A. D. A.* (with, following recent developments, the addition of *D. Cosmos*). The *American Dental Association Bulletin* will be *A. D. A. Bul.* This exception is mainly a matter of simplification, and is consistent with the method employed by the American Medical Association in their library work. There, the *Journal of the American Medical Association* is always abbreviated *J. A. M. A.*, while in other instances the form *Am.* is used. No misunderstanding develops in the case of the medical association, and in time no misunderstanding should develop from the use of *J. A. D. A.* in dental journalism. The two largest health professions in the country should be consistent in this simple usage.

2. *Association.* Both *Assoc.* and *Ass.* are now in use. However, the simple form *A.* seems to be readily understandable when in a combination. This form (*A.*) is used consistently by the *Index Medicus.*

3. *Bulletin.* The form *Bull.*, is regularly employed at present by the *Index Medicus* and the *Index-Catalogue.* The Wilson Indexes use the form proposed here, i.e., *Bul.*

4. *College.* While *Coll.* is frequently used, the form *Col.*, seems sufficient for interpretation.

5. *Dental* and *Dentistry.* Much confusion exists regarding the abbreviation of these two words. There is a rule which states, so the author was informed, that when there is a word of two or more syllables, the abbreviation is made up of the first three letters of the first syllable, and the first letter of the second. Since both these words are subject to that rule, the abbreviations would be the same.

Thus *Dent.* would be the abbreviation for both words, as is the case in most of the present usages. To avoid the resultant ambiguities when *Dent.* is used to designate both dental and dentistry, and to be consistent with the *Index Medicus* and *Index-Calalogue* uses of *M.* for medical and *Med.* for medicine, it is here proposed that *D.* be the abbreviation for dental, and *Den.* for dentistry.

6. *Journal.* The simplified form *J.* seems understandable in any combination that might be used. This is consistent with the *Index Medicus.*

7. *Quarterly.* Both *Quart.* and *Q.* are found in use. The form, *Quar.*, used in the Wilson Indexes, seems easy to interpret.

8. *Research.* This is frequently not abbreviated. However, the simple form, *Res.*, could be used with complete understanding.

9. *Transactions.* The shortened form *Tr.* is here preferred.

In addition, these general rules are suggested: (*a*) states of the Union be abbreviated as conventional usage decrees; (*b*) cities be abbreviated when usage permits as, Phila., Bost., San Fran., Cincin., Wash., Minneap., and Balt., and (*c*) state districts be reduced to the numeral in title abbreviations.

ILLUSTRATIVE USES

With this basic listing as a guide, the abbreviations have been applied to a few American dental periodicals which best show the usages suggested.

Annals of Dentistry	Ann. Den.
Bulletin of the Colorado State Dental Association	Bul. Colo. D. A
Bulletin of the Eastern Dental Society of Philadelphia	Bul. East. D. Soc. Phila.
Bulletin of the Second District Dental Society (New York)	Bul. 2nd Dist. D. Soc. (N. Y.)
Columbia Dental Review	Columbia D. Rev.
Dental Assistant	D. Asst.
Dental Hygiene Quarterly	D. Hyg. Quar.
Dental Outlook	D. Outlook
Harvard Dental Record	Harvard D. Rec.
Hudson County Dental Society Bulletin (New Jersey)	Hudson Co. D. Soc. Bul. (N. J.)
Illinois Dental Journal	Ill. D. J.
Iowa Dental Bulletin	Iowa D. Bul.
Journal of the American College of Dentists	J. Am. Col. Den.

Journal of the American Dental Association and Dental Cosmos	J. A. D. A. & D. Cosmos
Journal of the Canadian Dental Association	J. Canad. D. A.
Journal of Dental Education	J. D. Educ.
Journal of Dental Research	J. D. Res.
Journal of the Michigan State Dental Society	J. Mich. D. Soc.
Minneapolis District Dental Journal	Minneap. Dist. D. J.
Proceedings of the American Association of Dental Schools	Proc. Am. A. D. Schools
University of Illinois College of Dentistry Alumni Bulletin	Univ. Ill. Col. Den. Alumni Bul
Washington University Dental Journal	Wash. Univ. D. J.
International Journal of Orthodontia and Dentistry for Children	Internat. J. Orthodont. & Den. Children
Dental Survey	D. Survey
Nutrition and Dental Health	Nutrition & D. Health
Oral Hygiene	Oral Hyg.

For the attainment of uniformity, an aid is here suggested: that all dental periodicals *print* in some *conspicuous* place on their pages, the accepted abbreviation for their particular periodical. This might appear on the cover, the contents page, the mast-head, with the index, or in some such position, preferably beneath or near the full title. This would promote uniformity, and any investigator, editor, or librarian wishing to note a citation or reference would have the proper, accepted abbreviation at hand. A similar arrangement has been suggested, and is in use in certain European scientific publications.

REFERENCES AND CITATIONS

This is another phase of bibliographic method of importance to editors, and is included in this paper because of its close association with title abbreviations. There are two types: periodical citations, and book citations.

Periodical citations: The method proposed in this usage is consistent with that employed by the *Quarterly Cumulative Index Medicus*, and that suggested by Hackh (7). The reference gives, in the following order: 1—the name of the author; 2—the title of the article; 3—the abbreviation of the title of the periodical (italic); 4—the volume (bold-face type, Arabic numeral); 5—the paging; 6—the month (day of month if a semi-monthly, semi-weekly, or weekly publication), and 7—the year.

It should be noted that the title of the article is given in the original language if English, French, German, Spanish, Italian, or Portuguese, and in translation if in other languages. Also, that the title abbreviation is set in italic type; this impresses the reader as to location, and is accepted good form in most scientific publications. The volume number is in Arabic numerals instead of Roman; this conforms with modern usage, and has recently been adopted by the *Index-Catalogue*. The volume number is set in black or bold-face type. Another innovation concerns pagination: here there has been an obvious shortening; for example, pages 517–519 are given as 517–9; 384–398 as 384–98, while numbers such as 416–502 are given in full. The general order of the reference data is volume, page, month, and year, for example:

Jones, J. J. The physician and oral disease. *J. A. D. A. & D. Cosmos*, **25:** 118–25, Jan. 1938.

Deviations in usage will be noted from time to time. The title of the article is frequently not included. Occasionally the month is not mentioned. Notwithstanding these several variations, due mainly to editorial practices and format style, the essential order of the reference items should be maintained.

Book citations: In this instance reference has been made to the usages of the University of Chicago Press: 1—the name of the author; 2—the title of the book (italic); 3—the edition, if more than one; 4—place of publication; 5—publisher; 6—date, and 7—paging. Some departures from this outline necessarily will present themselves, but in general this order should be followed. It should be noted that the "facts of publication" are inclosed in parentheses; also, that these may be given in full or in partial form, but should appear in the same order. In the full form, a colon is used after place of publication, and commas are used elsewhere. When place and date of publication only form the statement, they appear in parentheses. The page reference, when unaccompanied by further details, must carry the abbreviation "p." or "pp." An example:

Jones, J. J., *Dental Histology and Embryology* (Philadelphia: P. Blakiston's Son & Co., 1938), pp. 167–72.

For more detailed information concerning references and citations, especially regarding the departures which are accepted, the reader is referred to the bibliography appended where the sources of these data may be studied.

Author's note: The findings contained in this paper served as the basis for the Report of the Committee on Abbreviations of the American Association of Dental Editors (1937). The work of this Committee is being continued, and their recommendations will not be adopted until the next annual meeting in 1938, when further study will have been completed.

Mainly, the continuation of the study is for the purpose of obtaining and evaluating the comments, criticisms, and suggestions of editors and interested individuals. Therefore, members of the International Association for Dental Research and other readers of the JOURNAL OF DENTAL RESEARCH are here afforded opportunity to coöperate with the Committee by discussing, with the Chairman, these points: List of Basic Abbreviations, Periodical Citations, and Book Citations. Thus, before a code of abbreviations is adopted by the American Association of Dental Editors, groups particularly involved will be able to check, study, and discuss the code details and related bibliographic data. Correspondence should be addressed to the author of this paper, in care of the School of Dentistry, University of Pittsburgh, Pittsburgh, Pa.

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INHIBITION IN VITRO OF DECALCIFICATION IN TEETH[1]

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In previous papers (3, 4, 5) the following conclusions were arrived at regarding decay of teeth:

1. There was a slight incidence of caries in pre-European Bantu and the most primitive of modern Bantu have an incidence of the same order (viz. about 30 per cent of all adults have caries with two or three carious teeth per carious mouth). 2. Those natives taking to a European mode of living show an alarming increase of caries which approaches or equals the tremendous incidence among Europeans of the present day. 3. Taking the major cause of decay first, and ignoring, for the time being, the reason for the decay of a trivial number of teeth in the primitive native, an attempt was made to correlate the presence of caries with various items of the diet. 4. This led to the conclusion that one or more of the following factors causes dental decay in those natives taking to a European manner of living (and hence in Europeans themselves): a. European bread; b. machine ground mealie meal; c. foods containing sugar. 5. It was argued that the crude cereal grains themselves were unlikely to be a major cause of caries since the primitive Bantu subsist on a very high cereal diet. 6. Similarly it was argued that sugar, as it occurs in nature, is not a major cause of caries. Natives in the sugar cane fields and factories chew cane regularly, and yet neither in this nor in other countries have they been found to be more prone to caries than their fellows. Fruits contain much sugar and they have never fallen under suspicion as producers of caries. 7. It was therefore argued that the cereals and sugars, as they occur in nature, are harmless, but that the refined states of these foods are dangerous to the teeth and produce caries. 8. To test this proposition by laboratory experiment, teeth were incubated in media containing sugar or cereals mixed with saliva. Refined white sugar produced decalcification in a high proportion of teeth, crude juice in very few. Similarly whole mealies and whole wheat produced this change in a significantly smaller proportion of teeth than the refined meal and flour made from these grains. 9. It is suggested that crude cane juice, whole mealie and whole wheat grains contain a "protective agent," which tends to inhibit the injurious action of the carbohydrate on the teeth. This "protective agent" is removed or reduced in concentration in the process of refining. It exerts its effect otherwise than by preventing or reducing the acid fermentation of the carbohydrate in the mouth.

Having been reassured of the existence in crude sugar and cereals of a "protective agent" which is removed or partially removed in the

[1] Most of the expenses of this work were defrayed by the Research Grant Board of the Union.

process of refining, the authors sought a clue to its identity. A thorough chemical and biochemical investigation of the difference between the natural and the refined states of these foods would involve years of careful work. Before attempting this, the authors decided to investigate the significance of some of the known differences.

The Nature of the "Protective Agent." Vitamin B stood out as a factor which is removed during the milling of cereals. The work of Peters et al. (6) has shown that Vitamin B_1 is concerned with the internal metabolism of carbohydrate, and that there is a tendency towards increased lactic acid in the blood and tissues in its absence. It seems possible that this factor may be involved here. This conception however, is too speculative for serious argument until more is known about the exact function of the vitamin and the actual metabolism of carbohydrates in the mouth.

A second possibility had more scientific backing. It is clear, from a consideration of simple physico-chemical theory, that the rate at which calcium phosphate dissolves is governed by two conditions: (a) the hydrogen ion concentration (the greater the acidity the faster the solution); (b) the product of the concentration of calcium and of phosphate ions (the greater this product the slower the rate of solution). The first is generally recognized and is the basis of the axiom that enamel will dissolve in an acid medium, and the greater the acidity the more it will dissolve. The second seems to have been overlooked.

Solid calcium phosphate in contact with an aqueous medium may be simply represented thus:

$$Ca_3(PO_4)_2 \rightleftharpoons 3Ca^{++} + 2PO_4^{---}$$

i.e., there is a constant conversion of solid material into ions and a constant conversion of ions back into the solid. (While the actual change is more complex than, this the additional complications do not affect the argument.) In neutral media the proportion of ions is infinitesimal, but as the medium is made more acid, the change from left to right is speeded up, with the result that more salt dissolves. By the mass-action law, the rate of change from ions to solid is proportional to the product (concentration Ca^{++})^3 $\times$ (concentration PO_4^{--}).^{2} For a given strength of acid, i.e., for a given pH, the

change from left to right will be constant; but the change right to left will be fast if the acid contains many calcium and phosphate ions, and slow, if it contains few or none. If the acid brings these ions with it therefore, the end result will be a slower rate of solution of the solid, for a constant strength of acid.

Both the refined and the unrefined sugar and cereals reached approximately the same acidity when incubated with saliva. It is known that crude cane juice contains both phosphorus and calcium, and that refined sugar contains no more than a trace of these elements; and it is also known that the milling and refining of flour and mealie meal reduces their calcium and phosphorus contents. It seemed plausible that the refined substances produced more decalcification than the crude (in spite of a constant pH) because the refined contained less of these elements and, therefore, presumably less of Ca and PO_4 ions than the crude.

EXPERIMENTAL

It was decided to determine what bearing this hypothesis had upon the *in vitro* decalcification of teeth. The procedure was identical with that used in previous experiments(5). A small quantity (0.5%) of $CaHPO_4$ was added to the media in a series of experiments. The results of these are recorded in Groups A, B and C in Table I. As in the earlier experiments, in each group teeth from the same subjects were used in the control and experimental tubes. The method having been explained by the data in the previous tables, details of individual experiments are omitted from Table I. It is clear that the addition of this quantity of $CaHPO_4$ gave no significant protection. The inference was that the concentration of Ca and PO_4 ions achieved in this way, was not sufficient to inhibit materially the destructive action of the medium.

The work of Robison and his colleagues on the normal calcification of bone (1, 2, 7) suggested a means of augmenting the concentration of these ions. Much of the phosphate in the blood and tissue fluids exists in combination with hexose sugars. This is not ionisable and therefore does not precipitate when it meets calcium ions. In certain localities in the body, especially in calcifying bones and teeth, there is phosphatase, an enzyme that hydrolyses the hexose phosphates,

TABLE I

*Effect of various substances on in vitro decalcification of teeth**

GROUP	SUBSTANCES CHEWED	NUMBER OF TEETH	NUMBER DECALCIFIED
A	Sucrose	10	6
	Sucrose & 0.5% $CaHPO_4$	8	4
B	Flour	12	11
	Flour & 0.5% $CaHPO_4$	12	7
C	Mealie Meal	12	9
	Mealie Meal & 0.5% $CaHPO_4$	12	8
D	Sucrose	10	8
	Sucrose & 2.0% Ca Lact. & 2.0% Na. Glycerophosphate	10	0
E	Sucrose	20	15
	Sucrose & 1.0% Ca. Lact. & 1.0% Na. Glycerophosphate	19	0
F	Flour	19	17
	Flour & 1.0% Ca. Lact. & 1.0% Na. Glycerophosphate	19	9
G	Mealie Meal	12	9
	Mealie Meal & 1.0% Ca. Lact. & 1.0% Na. Glycerophosphate	11	2
H	Sucrose	30	21
	Sucrose & 1.0% Ca. Glycerophos.	28	4
I	Flour	44	34
	Flour & 1.0% Ca. Glycerophos.	43	4
J	Flour	28	18
	Flour & 0.5% Ca. Glycerophos.	28	4
	Flour & 0.1% Ca. Glycerophos.	31	13
K	Mealie Meal	12	9
	Mealie Meal & 1.0% Ca. Glycerophos.	11	1

* Comparisons are only valid within each group.

giving ionisable inorganic phosphate. As calcium ions are always present, this creates a local condition of supersaturation of calcium

phosphate which tends to precipitate. Precipitation in supersaturated solutions is facilitated by the presence of the solid; the excess Ca and PO_4 ions precipitate most readily on preformed deposit of calcium phosphate. A rough qualitative test showed that saliva possessed the faculty of hydrolysing the glycerophosphates (which Robison found to be interchangeable with the hexose phosphates in his experiments).

Guided by this information, and with a view to creating a supersaturated solution of calcium phosphate in their decalcifying media, the authors introduced calcium lactate and sodium glycerophosphate. The results recorded in Groups D, E, F and G of Table I show that this produced considerable protection against decalcification. As a variant of the same experiment, calcium glycerophosphate was used with even better protection (Groups H, I, and K of Table I). Group J shows that in 0.5% concentration the salt gave good protection, but that the effect of 0.1% was so slight as to be of doubtful significance. Numerous pH estimations, in the course of these experiments, showed that a pH of about 4 to 5 was reached in all tubes within 24 hours and maintained until the end of the experiment.

In this search for the hypothetical "protective agent" of crude sugar and cereals, the authors have found a class of compounds which does reduce the decalcifying action of refined sugar, flour or mealie meal plus saliva *in vitro*. It has this in common with the "protective agent" of these crude foods—it exerts its effect otherwise than by controlling the pH. Whether the similarity extends further than this, and whether any practical use can be made of these organic phosphates in the control of dental caries, are questions which will require further investigation before they can be answered.

SUMMARY

1. By incubation in media containing refined sugar or cereals mixed with saliva decalcification was produced in healthy teeth. Calcium acid phosphate added to the incubating mixtures gave very little if any protection. 2. A mixture of calcium lactate and sodium glycerophosphate added to these decalcifying mixtures produced a marked reduction in the number of teeth attacked. 3. Calcium glycerophosphate was even more effective as a protective agent. 4. In none

of the tubes was the pH significantly different from the controls; therefore these protective substances, like the "protective agent" postulated for crude cane juice and cereals, do not exert their effect by controlling pH.

The authors wish to thank Dr. Monk and the staff of the Dental School of the University of the Witwatersrand, and the leading anesthetists of this town and their nurses, for supplying them with extracted teeth; also the subjects who supplied the saliva; and Mr. James Morris for his painstaking assistance at every stage of the work.

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A FURTHER CONTRIBUTION TO MANDIBULAR KINETICS

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This papers deals with two questions, (*a*) the relation between the masticatory movements in ruminants and in man and (*b*) the physiological rest-position of the mandible and its relation to the masticatory curve.

A. The relation between the masticatory movements in ruminants and in man

In my work published in 1931 on the masticatory movements in in man (1), the movements were regarded mainly from the kinematic standpoint. For reasons indicated it seemed convenient to make this limitation and, at the same time, propound the view that a comparison of the masticatory movements in man and in certain animals would undoubtedly be of great value. For widening our knowledge of the human mandible, we can study the evolution of its joint. If the view is accepted that a given function of an organ presupposes a certain construction of that organ, a view particularly justifiable in the case of a movement—a function which depends on the joint-surfaces, capsule, muscles, etc.—then a study of the masticatory movements in various animals may cast new light on the joint and its eventual development. One should distinguish between the masticatory movement proper, a function peculiar to mammals, and the seizing of the food which is common to all gnathostomes. In the lower gnathostomes the food is swallowed immediately after seizure but in mammals the seizing of food is followed by mastication, i.e., a more or less careful preparation of the food in the mouth prior to swallowing.

In the construction of the specialized joints in the higher mammals it is customary to distinguish between three different specialized masticatory movements, the hinge or opening and closing movement

in beasts of prey, the grinding and lateral movement in ruminants and the to-and-fro or gnawing movement in rodents. Animals employing all these movements in mastication are said to have a universal masticatory movement, e.g., swine, monkeys and man. The hyrax, a small kinsman of the elephant usually classed among hoofed animals although formerly considered among the rodents, exemplifies the universal mandibular function of the higher mammals—a peculiar combination of the ruminant, rodent and predatory movements. According to Lubosch (2) this animal chews its food to the accompaniment of lively, vibratory movements, the mandible is moved up and down, back and forth and from side to side. It should be observed that these movements are quite separate and not intermingled as in man. The lateral part of the condyle in the hyrax is human-like, the medial narrow and cylindrical, the glenoid fossa is shallow with strong medial inclination, the t. articulare low and the anterior teeth are of constant growth.

In the marsupials, lower mammals, similar evolutionary phenomena occur. Here the original type is a primitive form with a universal joint. Specializing has occurred in three different directions. Thus in the *Dasyuridae* or predatory marsupials there is a specialized hinge joint with a clearly developed t. articulare and cylindrical condyles on a level with the occlusal plane. Long canines tend still further to limit the range of movement. The kangaroo, one of the *Phalangeridae*, is furnished with a typical ungulate joint with a flat condyle and a very shallow fossa. The condyle is here considerably above the occlusal plane. The rodent type is also represented among the marsupials, e.g., in the *Didelphyidae* or pouched rats.

In mammals, especially the higher types, there is a formation in the mandibular joint, the meniscus articularis, which gives it a definite character. It is met with as early as the close of the Trias period. It is lacking in the sewer animals and some edentates, e.g. sloths and some marsupials. A retrograde formation also occurs in some of the higher mammals particularly those with specialized hinge-joint. The external pterygoid has not the same important function here as in the other two kinds of specialized movement—to draw the condyle forward so that the meniscus either becomes shrunk or is quite lacking after coalescence with one of the joint-surfaces.

It should be of some interest to compare the specialized masticatory movement in the three mammalian types with the movements of the human mandible. In these investigations of the ruminants and man roentgen kymography and kinematography have been employed. In studying the ruminants Lubosch (2) employed the subjective method of direct observation. *Figs. 1, 2 and 3* show some of Lubosch's curves depicting the movement as seen from the front, perhaps too regular. Direct observation may be employed for studying the periodicity of the movement as well as the general character of the preparatory movement. For example, the camel and the alpaca

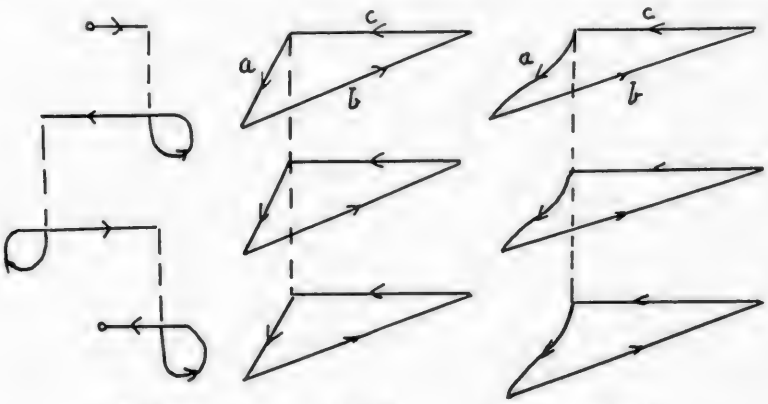

FIG. 1 FIG. 2 FIG. 3

seem to move the mandible in a circle (*fig. 1*) prior to the grinding movement proper and the antelope (*fig. 2*) and the buffalo (*fig. 3*) shift the mandible to the other side to get the movement well started but for exact reproduction of the movement direct observations should be rejected.

METHOD

Of the two methods employed for studying the movements of the lower jaw, for obvious reasons roentgen kymography is not suitable in the case of ruminants, kinematography being the only available method. An Agfa kino-camera giving 16 pictures per second was used. The duration of the masticatory periods varied from a half to a whole second per period, the goat giving 8 to 9 pictures per period; the stag 10 pictures per period; the buffalo and cow 12 to 14 pictures per period. For the graphic representation of a period it was found desirable to have about 15 pictures. A camera taking 32 pictures

per second would be ideal, a more rapid camera undesirable. In order to take the photographs without unduly disturbing the animals a teleobjective, focus width 5 cm., luminosity 1:3.5, was used. In recording, one picture at a time was projected onto a paper; a few well marked points on the head were taken as fixed points; the outlines of the mandible were drawn on transparent paper and a point was marked whose displacement thus formed the curve. The stag was the best experimental object, as it kept still for a number of periods and its head was the easiest to draw. The goat, buffalo and cow were less desirable subjects. Only those periods were recorded when the movement was carried back to the starting point.

OBSERVATIONS AND CONCLUSIONS

Stag (Cervus elaphus) chewing leaves. Mastication consists of a long repetition of identical movements. The movement is performed first four or five times on one side, then as many times on

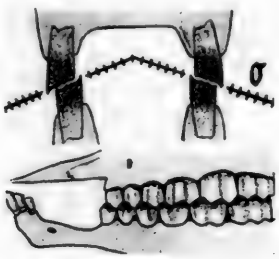

FIG. 4

the other side, then repeated. The number of chewings varies somewhat, but the total number of movements on either side appears to be constant. The type of this movement, seen frontally, appears from *fig. 5*. Six successive periods are depicted in *fig. 6*. Each period took $\frac{10}{16}$ seconds, the periodicity being well balanced. *Goat (Capra hircus) chewing cud.* The type of this movement appears from *fig. 7*. Several successive periods are depicted in *fig. 8*. A comparison of these curves in the stag and goat, kinematographically recorded, with the curves in *fig. 2* depicting the masticatory movements of the antelope from direct observations recorded by Lubosch (2), reveals little agreement. The grinding movement (*c*) cannot possibly be horizontal in view of the lateral downward inclination of the occlusal surfaces [*fig. 4, 0* from Gysi (3)]. Moreover, the opening movement is not carried so far laterally and, finally, the transition from

one movement to another is more gentle than is indicated by the abrupt curves of Lubosch.

Bison (*Bison bonasus*) *chewing leaves.* Two periods are recorded in *fig. 9*. *Neat cattle* (*Bos taurus*) *chewing grass.* Two periods are

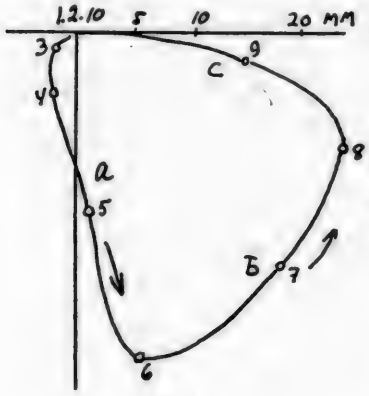

FIG. 5

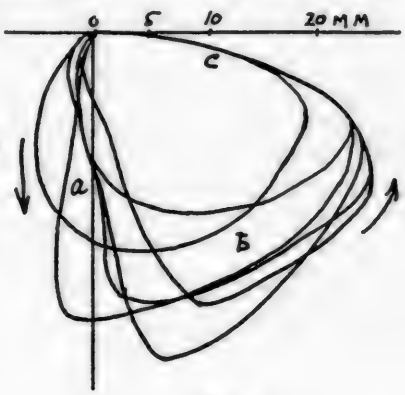

FIG. 6

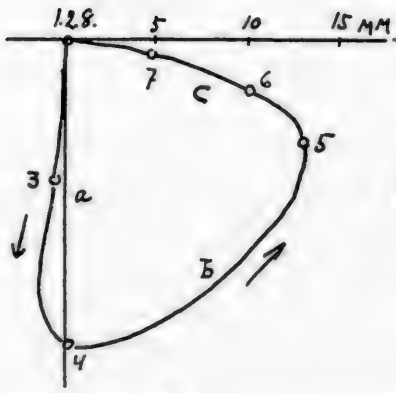

FIG. 7

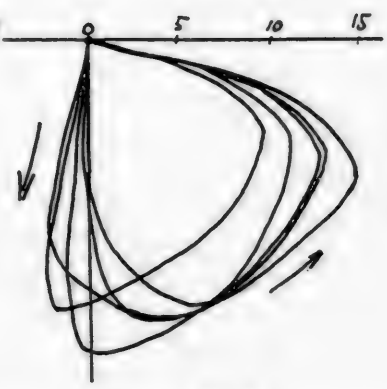

FIG. 8

recorded in *fig. 10*. The curves recorded (*figs. 9 and 10*) for the bison and the ox should be compared with *fig. 3* in which Lubosch records the movements of the buffalo. Here there is somewhat greater agreement than in the preceding group of animals. Apart from the

erroneous record of the grinding movement, the opening movement is well depicted on the opposite side to that on which the grinding movement is performed. The slight swing perceptible in the first part of the opening movement is noteworthy. In all four animals the curves can be distinguished as opening movement (*fig. 5a*), preparatory movement (*b*) and grinding movement (*c*). The opening movement is mainly straight down, but in the bison there is an evident swing-over to the side opposite the one where grinding is performed to give the mandible, as it were, more force for the preparatory movement. This movement might be called a "sling" movement. In the bison and the cow the opening movement takes

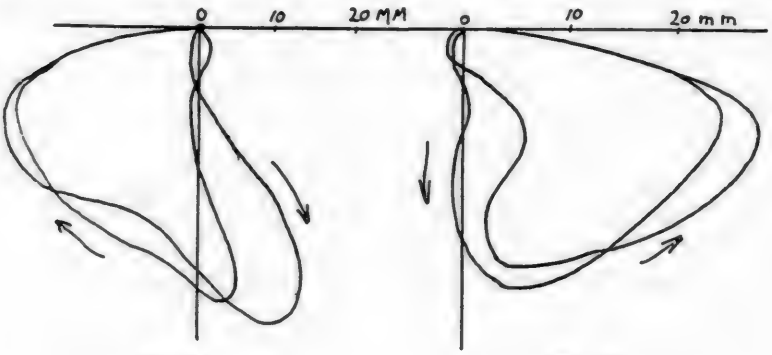

FIG. 9

FIG. 10

the shape of an S-curve while in the deer and goat it goes straight down.

The preparatory movement is practically identical in all four animals but most clearly expressed in the bison. The grinding movement is always the same in an individual, but its length may vary in different periods. The angle at which the grinding movement meets the horizontal plane varies in the four species, due to the differing inclination of the occlusal surface (*fig. 4, O*). This angle appears to be greatest in the goat.

If we now examine the curves of the movements of the human mandible (*fig. 11*) we very often find the individual movement divided into opening, preparatory and grinding movements and the individual

periods fairly similar but less specialized than in ruminants. The linked line represents the opening movement, the continuous line the closing movement, curve (*a*) the lateral gliding movement in a case of normal bite in chewing hard bread. The preparatory or "sling" movement, as one might call it, seems to lose itself half way and pass into a faintly specialized grinding movement. Occasionally the preparatory movement is found to be of a pronounced "sling" type as *fig. 12* indicates. The mandible is in a case of mandibular protrusion, chewing tough meat, brought immediately over to one side as if to gain more force for being slung over to the other side.

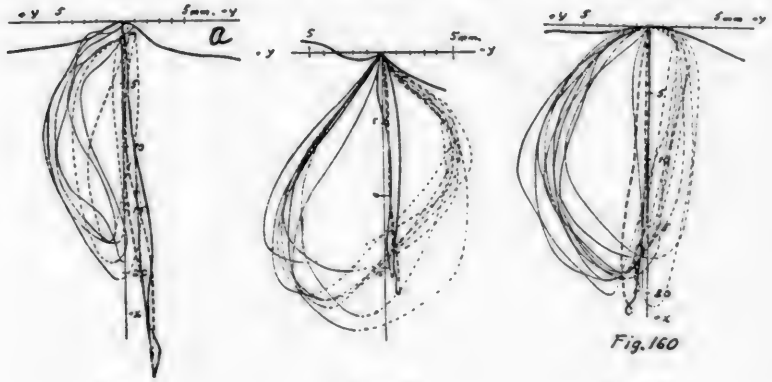

FIG. 11 FIG. 12 FIG. 13

(Compare movement in bison, *fig. 9.*) The human movement most akin to the ruminatory one is found in a case of labidodonti (straight bite) *fig. 13.* The preparatory movement is like that of the stag and the goat though the grinding movement is not so much employed.

It has been sought to show that human masticatory movements are to some extent reminiscent of the specialized movements in the ruminants. Owing to the universal character of the human masticatory movements there is reason to believe that the human mandible, while functioning, can perform movements which not only remind us of the ruminants but also of the other two groups of animals with specialized movements, viz., beasts of prey and rodents. Experiments already completed show that this assumption is justified.

B. The physiological rest-position of the lower jaw and its connection with the masticatory curve

Schwarz (4) made a fairly minute study of the relation of the position of the mandible to that of the head and in connection therewith the physiological rest-position of the lower jaw. When the head is held upright, the ear-nose plane being approximately horizontal, the lower denture is separated from the upper by a few millimetres. The condyle is at the bottom of the fossa but not in the maximal dorsal position. If the head is now bent backwards, the ear-nose

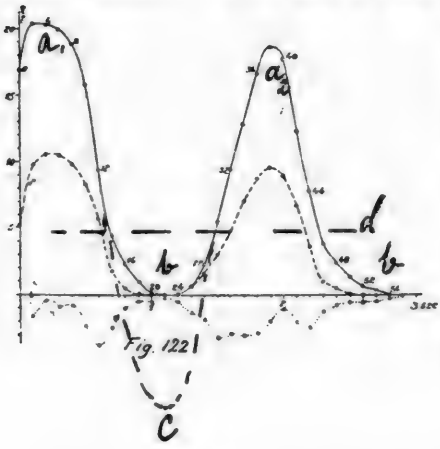

FIG. 14

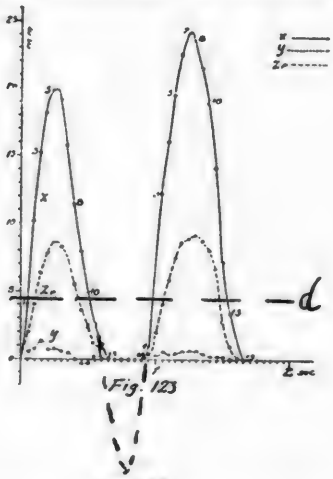

FIG. 15

plane sloping downward and backward and the lower jaw remaining slack, the lower denture is lowered yet more in relation to the upper, while the condyle glides toward the dorsal wall of the fossa. If, however, the head assumes a moderately ventral position with the Frankfort plane horizontal the lower denture approaches the upper and the mandible glides forward. If the head has a sufficiently ventral inclination, the incisors of the lower jaw will, in case of overbite, impinge upon the upper incisors.

In these studies of the masticatory movements, the experimental objects were placed so that Camper's (ear-nose) plane was horizontal. *Fig. 14* depicts a masticatory curve in mastication of tough meat

with the three specialized movements separately recorded. The abscissa denotes the time and the ordinate the path, the continuous line the opening and closing (predacious movement), the linked line the to-and-fro rodent movement, and the dotted line the lateral or ruminatory movement. A study of this curve shows that the apices (*a*) are more pointed than the bases (*b*). If the resistance, i.e., the upper denture and the food located at and immediately above the abscissa axis, were eliminated the momentum of the mandible would carry it along an imaginary curve down to *c* and then swing back to the position a_2. A sinus curve is then obtained whose axis would lie approximately at line *d* where the rate of movement appears to be greatest. This sinus curve evidently represents a harmonic swing-movement around the rest-position (*d*). If the experimental object now keeps the lower jaw slack, it will take up a position exactly corresponding to line *d*. The mandible may thus be said to describe during the masticatory function a harmonic swing-movement with its equilibrium coinciding with the physiological rest-position. *Fig. 15*, an opening and closing movement with empty mouth, shows the same thing.

I wish to express my cordial thanks to Professor G. Thouren for valuable advice. I am greatly obliged to the Warden of the Skansen Museum, Mr. A. Behm, for his kind permission to photograph the animals.

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SUPPLEMENT

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[2] During 1936–37 (July 1–June 30) meetings of sections were held on this schedule (compiled from reports of sectional secretaries, some of whom failed to respond or presented incomplete data): *Ann Arbor*—May 12. *Baltimore*—Feb. 1. *Boston*—Jan. 18, Mar. 25. *Chengtu*—Mar. 25, May 27. *Chicago*—Nov. 20, Jan. 22, May 21. *Cleveland*—Feb. 26. *Columbus*—Mar. 5. *Iowa City*—Dec. 4, Mar. 8. *London*—Oct. 14, Feb. 3, Apr. 14. *Louisville*—Apr. 6. *Minnesota*—Dec. 15, Apr. 15, June 15. *New York*—Jan. 28, June 3. *Philadelphia*—May 14. *Pittsburg*—Oct. 19, Jan. 11, Mar. 1, May 3. *Richmond*—Nov. 24, Feb. 17, Apr. 6. *Rochester*—Oct. 6, Jan. 6, Feb. 25, May 15. *St. Louis*—Nov. 10, Jan. 12, Apr. 13. *San Francisco*—July 13, Mar. 17. *Vienna*—Apr. 20. *Washington*—Nov. 30, Feb. 2, Apr. 13. *Winnipeg*—Oct. 12, Jan. 26, Mar. 8.

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21. *Prague Section.*—Karel Černý, Karl Häupl, Ján Jesenský, František Kostečka, Jaromir Křečan, A. E. Loos, V. F. Náprstek, **Friedrich Neumann*, František Neuwirt, Čestimir Parma, Josef Přibyl, Ferdinand Škaloud, Karel Wachsmann, Jr., Karel Wachsmann, Sr., Hans Wermuth.—15

22. *Richmond Section.*—Harry Bear, O. W. Clough, J. C. Forbes, W. B. Gurley, J. F. Hall, A. P. Little, Harry Lyons, **Grant Van Huysen*, J. B. Williams.—9

23. *Rochester Section.*—B. G. Bibby, R. E. Brawley, V. D. Cheyne, H. C. Hodge, Paul Lalonde, M. L. LeFevre, M. W. McCrea, **R. S. Manly*, H. Jobe Sedwick, R. S. Voorhees.—10

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26. *Toronto Section.*—H. K. Box, A. J. Broughton, Thomas Cowling, **A. W. Ellis*, G. W. Grieve, Andrew Hunter, F. C. Husband, A. J. McDonagh, E. W. Paul, H. S. Thomson.—10

27. *Vienna Section.*—Arwed Berg, Karl Breitner, **Fritz Driak*, Bernhard Gottlieb, Richard Grohs, Otto Hofer, Ernst Kellner, Emmerich Kotanyi, Hermann Mathis, Balint Orban, Hans Pichler, Otto Preissecker, Franz Schönbauer, A. M. Schwarz, Harry Sicher, Karl Spring, Georg Stein, Richard Trauner, Hermann Wolf, Josef Weinmann.—20

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29. *Winnipeg Section.*—**E. R. Bier*, F. T. Cadham, M. H. Garvin, K. M. Johnson, H. J. Merkeley, W. J. Riley.—6

30. *Not now members of a section.—Amsterdam.*—T. E. de Jonge-Cohen, Elkan Sanders. *Boston.*—Benjamin Tishler. *Buffalo.*—Mathew Podolin. *Denver.*—K. K. Cross. *Germany.*—Gustav Korkhaus, Friedrich Proell, Rudolf Weber. *Hartford.*—R. L. Coleman. *Johannesburg.*—S. F. Gottlich. *Liverpool.*—G. G. Macphee. *Milan.*—Gyula Csernyei, Silvio Palazzi. *Milford.*—Aaron Isaacs. *Montevideo.*—F. M. Pucci. *New York.*—H. W. Gillett. *New Zealand.*—C. D. M. Day, R. E. T. Hewat, R. M. S. Taylor. *Pittsburgh.*—Faith P. Hadley. *San Francisco.*—E. H. Mauk, F. V. Simonton. *Saranac Lake.*—W. E. Taylor. *Seoul.*—John L. Boots. *Swarthmore.*—J. A. Detlefsen. *Tucson.*—Margaret C. Smith. *Washington, D. C.*—Henry Klein. *Zurich.*—Walter Hess.—28

31. *Addresses as of Dec. 1 unknown.*—Guido Fischer, Martha R. Jones, Anna C. Nichols.—3

Total number of members—438; average membership of 29 sections—14.

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SPECIAL ABBREVIATIONS: *A. A. D. S.* = American Association of Dental Schools. *A. D. A.* = American Dental Association. *I. A. D. R.* = International Association for Dental Research. *J. D. R.* = Journal of Dental Research.

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INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Organized: New York City, Dec. 10, 1920. Of those at the meeting of organization, the following remain active members: Adolph Berger, Theodor Blum, G. W. Clapp, H. S. Dunning, W. J. Gies, H. W. Gillett, R. G. Hutchinson, Jr., T. P. Hyatt, Milo Hellman, F. C. Kemple, A. H. Merritt, B. B. Palmer, P. R. Stillman, L. M. Waugh.

General Meetings have been held on this schedule: (1) New York, Dec. 21, '22. (2) Chicago, Mar. 7, '24. (3) Chicago, Mar. 20, '25. (4) Chicago, Mar. 26, '26. (5) New York, Apr. 19, '27. (6) Washington, Mar. 25–28, '28. (7) Chicago, Mar. 23–24, '29. (8) Toronto, Mar. 24–26, '30. (9) Memphis, Mar. 21–22, '31. (10) Columbus, Mar. 19–20, '32. (11) Chicago, Mar. 18–19, '33; adjourned executive session, New York, May 25, '33. (12) Chicago, Mar. 17–18, '34. (13) Chicago, Mar. 16–17, '35. (14) Louisville, Mar. 14–15, '36. (15) Baltimore, Mar. 13–14, '37.

Objects (Const., Art. II): . . . "(a) To promote broadly the advancement of active research in all branches of dental science and in related phases of the sciences that contribute directly to the development of dentistry, and which add to the knowledge of the mouth and teeth and of their relations to the body as a whole; . . . (b) to encourage and facilitate coöperative effort and achievement by, and mutual helpfulness among, investigators in all nations in every division of stomatology; (c) to the end, particularly, that dentistry may render cumulatively more perfect service to humanity." (The Association has been using "dentistry" and "stomatology" as synonyms.)

Membership: total (June 30, 1937), 439. *Eligibility (Const., Art. 14, Sec. 1).*—"Any person who has conducted, and published an account of, a meritorious original investigation in dental science, or in any of the sciences contributory to stomatology, shall be eligible to membership in this Association, providing such person conforms to the recognized standards of professional ethics." *Election.*—New members may be elected *only at general meetings of the Association.* Membership in a section is secondary to membership in the Association as a whole. Removal of members from one geographic center to another does not affect their membership in the Association.

Endowment fund (as of March 5, 1937): $1,205.72. There have been no expenditures from this fund.

Divisions may be organized by the members in any nation. There are now divisions in Austria, Canada, China, Czechoslovakia, England, Hungary, South Africa, and the United States—8. *Sections* may be organized by the members in geographic centers within the divisions. There are now sections (27) in Vienna ('29); Halifax ('28), Toronto ('21), Winnipeg ('30); Chengtu ('34); Prague ('32); London ('31); Budapest ('34); Johannesburg ('34); Ann Arbor ('23), Baltimore ('33), Boston ('20), Chicago ('20), Cleveland ('30), Columbus ('32), Detroit ('37), Iowa City ('36), Louisville ('32), Minnesota ('28), New Haven ('30), New Orleans ('37), New York ('20), Philadelphia ('28), Pittsburgh ('28), Richmond ('33), Rochester ('33), St. Louis ('28), San Francisco ('24), Washington, D. C. ('31). The names of the secretaries are given on p. 2 of this cover.

Sectional meetings in 1936–37. Ann Arbor—May 12. *Baltimore*—Feb. 1. *Boston*—Jan. 18, Mar. 25. *Chengtu*—Mar. 25, May 27. *Chicago*—Nov. 20, Jan. 22, May 21. *Cleveland*—Feb. 26. *Columbus*—Mar. 5. *Iowa City*—Dec. 4, Mar. 8. *London*—Oct. 14, Feb. 3, Apr. 14. *Louisville*—Apr. 6. *Minnesota*—Dec. 15, Apr. 15, June 15. *New York*—Jan. 28, June 3. *Philadelphia*—May 14. *Pittsburgh*—Oct. 19, Jan. 11, Mar. 1, May 3. *Richmond*—Nov. 24, Feb. 17, Apr. 6. *Rochester*—Oct. 6, Jan. 6, Feb. 25, May 15. *St. Louis*—Nov. 10, Jan. 12, Apr. 13. *San Francisco*—July 13, Mar. 17. *Vienna*—Apr. 20. *Washington*—Nov. 30, Feb. 2, Apr. 13. *Winnipeg*—Oct. 12, Jan. 26, Mar. 8.

Journal of Dental Research: now the Association's official publication. See register of officers and editors on page 2 of cover. Bi-monthly issue has been resumed with this volume.

Sixteenth General Meeting of the International Association for Dental Research

The next general meeting of the International Association for Dental Research will be held in Minneapolis on March 12–13, 1938, in coordination with the meeting of the American Association of Dental Schools. Headquarters will be Hotel Nicollet.

Resolution Adopted by the International Association for Dental Research Relating to Proprietary Dental Journals, Louisville, Ky., March 15, 1936

Whereas, it is the consensus of opinion of our members that association, either as a contributor or as a member of the editorial staff, with proprietary publications that are distributed free of charge to the members of the dental profession—and whose chief object is the advertisement of commercial products—is undesirable; therefore be it *Resolved* that the International Association for Dental Research disapproves such association by its members, and by applicants for membership in the Association.

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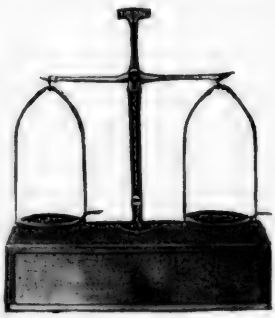

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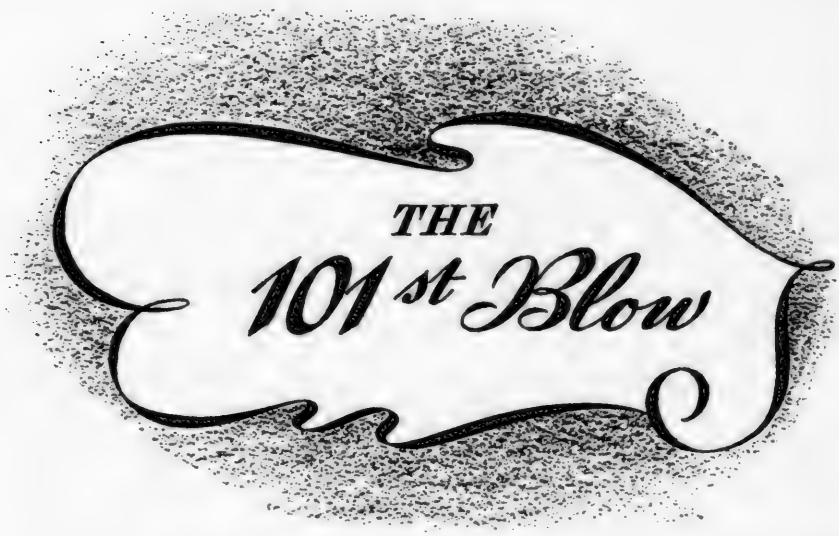

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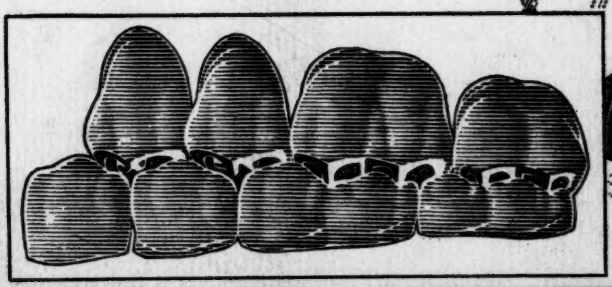

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